PRACTICAL MANUAL

OF THE

DISEASES OF THE HEART

AND

GREAT VESSELS.

A WORK INTENDED TO FACILITATE AND EXTEND THE STUDY OF THESE DISEASES.

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PREFACE.

Notwithstanding the publication of many valuable treatises on the diseases of the heart, their study is generally neglected.

The unfounded skepticism which still exists in the minds of many physicians, on the possibility of recognising and curing these diseases, and, especially, the differences of opinion among authors respecting them, have, in no slight degree, contributed to cast disfavour on this useful branch of medicine. Struck with these differences when we began to devote ourselves to the study of cardiac diseases, it seemed to us, that the best means of acquiring a just idea of the whole subject would be, to combine all the materials furnished by authors, and to test by clinical observation the truth of their statements.

This is the undertaking, these are the investigations in which we engaged, for our own satisfaction in the first place, and which we now offer to the public. The treatises of Senac, of Corvisart, of Laennec, of Bertin, of M. Bouillaud, of M. Gendrin, and especially the excellent work of an observer whom death has prematurely removed from the field of science (Dr. Hope), have been our principal guides. We have not adopted servilely the opinions of any one. We make no pretensions to novelty; but we have compared, and, as well as we have been able, have verified, for ourselves, the observations of others.

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A MANUAL

ON

DISEASES OF THE HEART,

ETC., ETC.

FIRST PART.

ANATOMY AND PHYSIOLOGY OF THE HEART.

CHAPTER I.

THE ANATOMY OF THE HEART.

THERE is at present no truth better established in medicine, than that of the indispensable necessity of studying perfectly all the phenomena, and all the conditions appertaining to the healthy state of the organs, in order to understand the different modifications which they and their functions undergo during their morbid states.

It is only by comparing the pathological transformations with what experience has taught us to be incident to the normal state, that we can hazard an assertion respecting the nature of any lesion or of any modification of function.

The perfect knowledge of the anatomy and physiology of the heart in its healthy state, is therefore indispensable to the study of the diseases and pathological alterations of this organ.

It is not our intention to introduce here the details of descriptive anatomy, which are readily met with in books on that subject; neither is it necessary to say anything concerning the anatomy of its texture, on which point the able works of Steno, Borelli, Wolf, Duncan, and Gerdy, leave nothing to be desired.

But there are some points, of which authors of topographical anatomy have, in general, treated very lightly, such as the exact position of the heart in its relation to the different parts surrounding it, its weight and size, and the relative volume of its different portions to each other.

It is well known, that the heart, being the central organ of the circulation, occupies in the chest the inferior portion of the anterior mediastinum; that it is enveloped by a fibro-serous sac, called the pericardium; that it rests upon the diaphragm; that it looks obliquely from above downwards, and from before backwards; and that, while its body and apex remain free, its base is fixed, whence are detached the large vessels.

It must be evident, from the preceding account, that the heart, resting upon the diaphragm, and being free at its apex, describes, at each movement of the diaphragm, an arc of a circle, of which the axis of the heart is the radius, and the base of that organ the centre. It would be impossible to appreciate the changes which take place in the several relations of this organ, if there was not a point of departure which remained invariable in its position; this fixed point is not exactly that which follows the movements of the diaphragm, but rather the point at which the large arteries are detached from the heart. Dr. Hope has shown, and M. Gendrin has confirmed it by his experiments, that this point, where the motion of the heart and the large vessels arising from it terminates, this axis of the movements, corresponds to the half of its extent, and to the centre of the pulmonary artery; that is to say, to the middle of the space embraced between the origin and the division of this artery, or rather to the middle of the space comprised between the synchondro-sternal articulations of the second and third left ribs.

At their origin, the two large arterial trunks which arise from the heart are placed above each other; the aorta, which is posterior, extends, by about one-third of its volume, beyond the border of the pulmonary artery, on the right side.

If we draw from one side to the other a horizontal line, along the inferior border of the two third ribs, this line passes over the valves of the pulmonary artery, which it leaves, situated a little to the left of the median line; the valves of the aorta are situated behind the preceding, but about half an inch lower, so that the artificial line just described, on one side, passes above the base of the valves of the pulmonary artery, whilst, on the other, it corresponds to the free extremity of the valves of the aorta. It is over the track of this line, and consequently on a level with the inferior border of the third rib, that the murmurs produced by the diseases of those valves, are found at their maximum. From this point the aorta and pulmonary artery arise; the pulmonary artery, at first, in contact with the sternum, afterwards inclines to the left, until it reaches the interval between the second and third ribs, where the fixed point of the movements, before alluded to, will be found. If, instead of forcing a needle through the second intercostal space, we force one immediately under the inferior edge of the synchondro-sternal articulation of the second rib, the needle traverses the pulmonary artery on a level with its bifurcation, and meets behind it the remains of the *ductus arteriosus*.

The aorta, inclining slightly to the right, goes afterwards from the right side to the left, by passing before the right branch of the pulmonary artery. It is then found in its relation with the sternum, at the moment when it disengages itself from beneath the pulmonary artery, that is, on a level with the superior border of the synchondro-sternal articulation of the second right rib.

It is also under this last articulation that the origin of the brachio-cephalic trunk is found. As regards the origins of the left carotid and the left subclavian arteries, they are found beneath the sternum, behind the synchondro-sternal articulation of the second left rib, and behind the left branch of the pulmonary artery.

The auriculo-ventricular orifices are situated in the space included between the third and fourth left ribs, near the sternum; that of the right ventricle is a little lower than that of the left ventricle.

The heart rests, by its flattened face, on the diaphragm; its point is inclined downwards, forwards, and to the left of the chest; its base looks upwards, backwards, and to the right; the left ventricle is posterior and superior; the right ventricle is anterior and inferior.

The apex of the heart generally pulsates between the cartilages of the fifth and the sixth left ribs, and sometimes between the cartilages of the fourth and fifth, at four or five centimetres,* or little more than an inch, from the left extremity of the sternum; in women and children, it beats oftener between the fourth and fifth left ribs.

The right auricle, which is situated before and to the right of the base of the heart, is partly concealed by the anterior border of the right lung; but its appendix and a part of its body correspond immediately to the sternum, or, more strictly speaking, to the synchondro-sternal articulation of the third rib. The left auricle, which is situated behind and to the left of the base of the heart, is covered, as well as its appendix, by the anterior border of the left lung, and corresponds to the interval between the third and the fourth ribs.

The vertical line through the synchondro-sternal articulations, divides the heart into two unequal parts, leaving one-third on the right, and two-thirds on the left side. The portion of the heart situated to the right, contains the superior extremity of the right ventricle, and the right auricle; that portion lying on the left side, contains the inferior part of the right ventricle, the left ventricle and auricle.

The pericardium has a pyriform appearance; enlarged at its middle, and extending, especially to the left, it continues to contract more in proportion as it approaches the point where it is reflected upon the large arteries which take their origin from the base of

* Centimetre is a French measure, equal to ·3937 of an inch.

the heart, that is, on a level with the synchondro-sternal articulation of the second left rib.

The lungs descend separately, for the extent of two inches, along the edges of the sternum; they cover the base of the heart slightly on the right side, but to a much greater extent on the left; from this place, they separate from each other; the left lung following the course of an oblique line, drawn from the internal end of the cartilage of the second rib to the anterior extremity of the last rib; the right lung following the track of an oblique line, drawn from the median and superior extremity of the sternum to the anterior extremity of the first false rib; and by this separation, they leave exposed a considerable portion of the right ventricle, and a much smaller portion of the inferior part of the left ventricle.

We are far from attaching, to the estimate of the volume and the weight of the heart, all the importance which some persons give to them; we are aware, that variable as they are, according to the subjects, they also vary in the same persons at different epochs of their life, and under very diversified influences; but we also think, that all sciences gain in point of precision and certainty, by establishing moderate limits, as standards, with which we can compare the alterations that are observed: we think, in conclusion, that the appreciation of the quantity must be left as little as possible to conjecture, because few persons judge rightly in regard to this point. We would have everything known, relative to the weight and volume of the heart; but we refer what is further to be said of this point, to the article Hypertrophy, where other data will be found more in place.

CHAPTER II.

PHYSIOLOGY OF THE BEATS AND THE SOUNDS OF THE HEART.

SECTION FIRST.

OF THE MOVEMENTS OF THE HEART.

It is only necessary to apply the hand over the præcordial region, to be sensible that the heart executes movements; the shock which is perceived in this region, and which constitutes what is called an *impulse*, can leave no doubt, in respect to this fact. The name of

systole has been given to the contraction of the cavities of the heart, and that of *diastole* to their dilatation.*

The fact is well known, that the blood arrives at the auricles through the large veins; that from the auricles it passes into the ventricles, whence it is projected into the pulmonary artery and aorta. But in what order, or in what succession do the contraction and dilatation of the cavities of the heart take place? According to Haller, the auricles contract simultaneously, whilst the ventricles are in a state of repose; and, when the auricles are in a state of relaxation, both ventricles contract simultaneously, like the auricles; to this last contraction succeeds a relaxation, which is a true state of rest; it is during this interval that fresh contractions of the auricles take place. As has been seen, Haller supposed that the contraction of the auricles was entirely independent of that of the ventricles, and that the contraction of the latter (the ventricles) only began after the relaxation of the auricles. M. Magendie has gone still further; he has admitted a species of antagonism, by virtue of which the auricles and ventricles contract and dilate alternately; so that the contraction of the auricles coincides with the dilatation of the ventricles, and the contraction of the ventricles with the dilatation of the auricles.

The beautiful experiments of Dr. Hope have evidently proved, (contrary to the opinion of Magendie,) that the series of movements of the heart takes place in the following manner:

The first movement of the heart, which interrupts the interval of repose, is the systole of the auricle; it consists in a slight and very short contractile movement, more marked towards the appendix than anywhere else, extending by a rapid, and as it were, vermicular motion towards the ventricle, and terminating in the systole of this ventricle, rather by continuity of action, than by two successive movements.

The systole of the ventricles begins suddenly; and, at the moment, when the auriculo-ventricular valves are shut, there is perceived, in their vicinity, a strong lateral impulsion; at the same time the parietes of the heart acquire great firmness; the apex is carried forwards and approaches the base; in other words, the vertical diameter of the heart is shortened, and its transverse diameter increased. Coincident with the systole, we have the impulse of the point of the heart against the ribs, the pulse, or the arterial diastole of the arteries, near the heart; and, finally, a peculiar sound, which

* Are the diastole and systole active movements of the heart? This question has been differently answered. There can be no doubt respecting the active character of the systole, which is accompanied in the heart by the same phenomena as belong to the contraction of the muscles of animal life: as to the diastole, it is difficult to admit its activity, since there is no disposition of fibres to cause it. It is, therefore, probable that it is the result of that power which belongs to muscles, and by virtue of which they pass from the state of relaxation to that of repose: it is simply a degree of elasticity.

will be investigated in another place, under the name of first, or systolic sound.

The systole of the ventricles is followed by their diastole, during which period they return by an instantaneous expansion, which is sensible, both to the touch and sight, to the same state in which they were found in the preceding interval of repose. The movement of the diastole is accompanied by the afflux of blood into the ventricles, from the auricles, by a slight retraction of the auricular cavities, by the removal of the apex of the heart from the parietes of the chest, and by a particular sound, of which we shall speak presently under the name of second, or diastolic sound.

Then succeeds the interval of repose; during which time, the ventricles remain in a sate of fulness, not amounting to distension. This interval measures the time which elapses between the second and first sound of the heart; the auricle only rests during the first portion of this interval, the remaining time being occupied by the contraction of its cavity, and with it recommences the whole series of movements we have just described.

The name of a complete beat, or revolution of the heart, is given to the combination of its movements, and state of repose, which constitute the action of this organ, extending from one systole to the succeeding systole. The heart accomplishes its revolution ordinarily in one second, and sometimes in less time in adults; the number of pulsations, supposing them to be only 60 per minute, amounts in one hour to 3,600, and in a day to 86,400.

According to Laennec, the respective duration of the different movements, which constitute a beat of the heart, is as follows: the systole of the ventricles occupies one-half, or thereabouts, of the whole period of a pulsation; the ventricular diastole occupies a fourth, or at most a third, of this period; the other fourth, or a little less time, belongs to the interval of repose. (It is in the last portion of this interval, that the systole of the auricles is placed.)

Mr. Bryan, who has measured by quite an ingenious process the duration of the different movements of the heart, has arrived at this conclusion; that the contractions of the ventricles do not occupy half of the duration of a pulsation, but only a little less than a third of this period.

M. Gendrin, introducing into the examination of this question all the accuracy of observation belonging to him, has found, in measuring by means of a watch with a second-hand, that the duration of the contraction of the ventricles, measured by the duration of the impulse of the apex of the heart against the parietes, is less than twenty-four "*tierces*," or less than two-fifths of a second; that the systolic contraction, the period of silence which follows, and the diastolic sound, take place in thirty "*tierces*," or half a second; that, consequently, during more than half a second, the heart, being relaxed, and allowing the blood to penetrate into its cavities, remains in a complete state of repose. The knowledge of the duration of the dif-

ferent motions of the heart is not a thing of mere curiosity, since it is by this knowledge that we can determine how long a time the different cavities of the organ are in action.

If the opinion of Laennec be received, the ventricles have twelve hours of repose out of the twenty-four, and the auricles have eighteen hours; whilst, according to Mr. Bryan, the ventricles would have sixteen to seventeen hours rest. Dr. Hope thinks, on the contrary, that they have only six hours rest; but it is proper here to remark, that he does not take into account the six hours which the diastole of the ventricles occupies, which he regards as not being a state of true repose.

As regards the auricles, this author requires, that the period of their repose should be equal to that of the ventricles. In accordance with the more correct estimation of M. Gendrin, the active and energetic contraction of the heart continues really only four or five hours during the day. This organ occupies, in the transition from the state of active contraction to that of complete repose, a period which is almost double that of the instantaneous initial contraction, or a little less than six hours; and hence, the complete repose lasts more than twelve hours. As to the repose of the auricles, M. Gendrin computes it at fifteen or twenty "*tierces*" out of a second (fifteen or sixteen hours a day).

If there exists a phenomenon, respecting the contraction of the heart, which has greatly interested physiologists, it is certainly the shock of the point of the heart against the præcordial region, or the *impulse* of this organ. It is now generally admitted, that this shock is produced during its systole, and also that it coincides with the systole of the ventricles; it is surprising to see distinguished men, as MM. Pigeaux and Beau, sustain a contrary opinion; for, by placing one hand on the præcordial region, and the other on the carotid, the fact is clearly proved, that the arterial impulse corresponds perfectly with the impulse of the extremity of this organ; again, it is only necessary to expose the heart of a living animal, to see that the point of the heart is constantly carried forwards during the systole, whilst, during the diastole, the heart experiences no displacement.

It is evident, that the cause of the impulse of the heart must be found in the blow of its apex against the thoracic parietes; but as the left ventricle most often forms this point, it follows, that the former is the principal agent of the impulse of this point against the chest; but should we conclude from this, as M. Filhos has, that the fibres of the right ventricle do not concur at all in the production of this phenomenon?

Without speaking of the disposition of these fibres, which is very far from being unfavourable to the production of this movement, facts prove that, in certain cases of hypertrophy of the right ventricle, the apex of the heart was formed entirely by this ventricle, and that the impulse in this case was not wanting.

But what is the immediate cause, or rather what are the essential

causes of the impulse of the heart against the thoracic parietes? It has been admitted for a sufficiently long time, with Senac, that the impulse of the apex of the heart depended, partly, on the dilatation of the aorta and pulmonary artery, and on the straightening of the arch of the aorta; partly on the reflux of blood, which takes place from the ventricles into the auricles, especially into the left auricle, at the moment when the auriculo-ventricular valves approach each other, which is, besides, an accessory cause of the considerable dilatation of the left auricle that lies pressed between the base of the ventricles and the dorsal vertebræ. Without noticing the difficulties which would prevent our understanding how the dilatation of the arteries arising from the heart, and the straightening of their curvatures, could cause the apex of the heart to move forwards; without discussing, also, the erroneous opinion advanced, that the aorta straightens itself during the influx of blood, since Gendrin and Carson have proved, that the curvature, instead of being diminished, is considerably augmented; without dwelling longer on the fact of comparative anatomy, noticed by Haller, that among certain animals, in whom the arteries which spring from the heart present no curvature, this organ is not the less carried forwards during the systole; it suffices, in order to show how defective is the first part of Senac's explanation, to remark here, that the movement forwards, which the apex of the heart executes, coincides perfectly with the systole of the ventricles. Whereas, according to the theory of Senac, this movement ought to take place after the systole; when certainly the reflux of blood which takes place into the auricles, and especially into the left auricle, at the moment of the systole of the ventricles, produces, however inconsiderable it may be, a superabundance in the auricles. The blood, therefore, which then suddenly distends the auricles, being thrown back on the fourth dorsal vertebræ, which serves as a support to these cavities, and principally to the left auricle, thrusts the heart forwards, with all the force with which the systole of the left ventricle acts upon it, in order to force it again into the left auricle. We admit this forced dilatation of the auricles as one of the elements in the production of the stroke of the apex of the heart against the walls of the chest; but only as a very feeble element, since the quantity of blood, returning into the auricles at each contraction of the ventricles, is by no means considerable, and the force with which the heart is carried forwards, is in a direct ratio with the quantity of this liquid returned.

There exists a fact, which settles this question, and which has been perfectly proved by Hope and Bouillaud; it is, that the forward movement of the point of the heart is almost entirely independent of the accessory conditions to which it has been wished to attach it; viz., that it is the result of a disposition, or a power of its muscular fibres. If we tear out the heart of a living animal, and place it on a table, deprived of both the power of straightening the curve of the aorta, and of causing the dilatation of the arterial trunks, &c., it continues, nevertheless, to contract, and each con-

traction is accompanied by an elevation of its apex. At the moment when the heart contracts, it becomes round, and rests on the diaphragm only, which before sustained it, by a very small point of contact; consequently the apex is elevated, and is more elevated as it is lighter and more easily moved than the base, and as this last is retained by the weight of blood in the auricles and large vessels, and also by its own weight; besides, the muscular fibres arising near the arterial circles, and consequently from the most anterior part of the heart, tilt the rounded body of the ventricles upon the sinuses of the auricles, which are placed at the most posterior part of the heart; the point of the heart is thereby carried very rapidly forwards, because, (if we may use the expression,) the apex of the heart represents the long arm of the lever, the auricles the fulcrum, and the power is found at the origin of the aorta and pulmonary artery; finally, in proportion as the ventricles contract, the apex of the heart is carried more and more upwards and forwards, as Senac had already remarked, in consequence of the distension which is incessantly increasing in the auricles.

This theory, which is due to Dr. Hope, appears to us to obviate all objections.

At this present time, M. Gendrin has shown that, among the elements in the production of this movement, on the one hand, the extension of the aorta and pulmonary artery, under the influence of the penetration of blood into their cavities, plays an important part; and on the other hand, that there is a kind of projection forwards, similar to a rebound, which the heart experiences when passing, by an instantaneous and truly convulsive movement, to the spherical form, it finds in the resistance of the diaphragm and in the body of the ventricles, a fixed point, which serves as a support. Of these two causes, the first does not appear sufficiently proved to be an element of this movement. Nothing is certainly better established, than the fact of the elongation and dilatation of the arterial tubes, by the sudden and forced introduction of blood into them; but we think that this elongation is more than compensated for by the shortening of the heart. How, indeed, can we admit this element, when experience, that judge of final appeal, has shown that, in the systole of the heart, its apex does not glide on the floor of the diaphragm, but, on the contrary, that it is carried from below upwards, and from behind forwards. As to the second cause, we only admit it as accessory, in consequence of the reasons already made known.

To resume: we are of opinion, that the impulse of the apex of the heart against the thoracic parietes is due principally, 1st, to the contraction of the circular fibres of the heart, which, by fixing their point of support on the arterial orifices, into which they are inserted, tilt the ventricles on the auricles, and carry the point of the heart strongly upwards and forwards; 2d, as an accessory cause to the movement of rebound which the heart experiences when it passes suddenly to the state of sphericity; 3d, to the reflux of a

certain quantity of blood into the auricles, and principally into the left auricle.

The systole of the ventricles has, for its object, to empty almost completely their cavities. We say *almost* completely, for there are some animals, (the frog, for example,) in which the transparency of the heart allows us to recognise the fact, that it is completely emptied; in large animals, on the contrary, it is impossible to assure ourselves of this fact, in a positive manner; and if we may judge, from the diminution of volume which the ventricles experience, it is more than probable that they are not entirely emptied. As to the auricles: they remain constantly in a state of fulness, and the extent of their contraction is so inconsiderable, that it does not amount to a third of their volume.

The diastole of the ventricles, which succeeds their systole, and whose office it is to allow a fresh quantity of blood to penetrate into the cavities, is not, as Laennec thought, determined by the contraction of the auricles; this contraction is too feeble and too limited for this to occur.

The diastole is effected by causes much more powerful in their operation; 1st, that particular force, (no matter whether elasticity or something else,) by virtue of which the heart passes from a state of contraction to that of relaxation, and by virtue of which, it exercises a suction power over the circulatory system; 2d, the distension of the auricles, which is at its maximum at the moment when the systole terminates; 3d, the weight of the ventricles, which presses on the distended auricles placed beneath them; 4th, the width of the auriculo-ventricular orifice, which permits the blood to enter freely into the ventricles; 5th, the feeble contraction of the auricle, or rather of the auricular appendix.

The systole and the diastole of the ventricles are accompanied, in the interior of the heart, by movements, which consist in the depression and straightening of the valves. The mechanism of their movements varies, according to the seat which they occupy; thus, the valves of the arterial orifice are entirely passive in their movements; they obey, in their elevation, the impulse of the column of blood which the heart projects during the systole, and in their depression, partly owing to the backward impulse of the column of blood, and partly the species of suction which the ventricles exert during the diastole. The valves, which occupy the auriculo-ventricular orifice, are only passive during their depression, yielding then to the weight of the sanguineous column which comes from the auricles, and to the suction exercised by the ventricles; but their tension or elevation is, on the contrary, entirely an active movement, like the ventricular systole, with which it coincides.

It only remains now to explain, in a concise manner, all the mechanism of that very complicated apparatus, called the heart. The best general idea that can be given, is to compare it to a double

suction and forcing pump, acting on the liquid, which is called the blood.

To explain this mechanism, let us admit that the ventricles have just contracted, and that the action of the heart commences with the diastole of the ventricles; while these ventricles pass from the state of contraction to that of relaxation, the blood, obeying all the powers which move it, penetrates through the auriculo-ventricular orifices, by depressing the mitral and tricuspid valves, and turning their extremities outwards. To this dilatation succeeds the interval of repose, during which time the ventricles remain in a state of fulness without distension, until the moment, when the auricles, in contracting, throw a fresh wave of blood into the ventricles, and when their contraction is propagated even to the ventricular cavities.

The contraction of the ventricles, pressing the blood on all sides, tends to drive it through the auriculo-ventricular and arterial orifices; but, at this moment, the mitral and tricuspid valves, sustained outside by the blood, which is present in the ventricle, and inside by the blood, which they force back into the auricles, being rendered tense, and raised besides by the fleshy columns of the heart, oppose the entrance of blood into the auricles; whilst the arterial valves, being raised up, permit the column of blood to pass. But, as soon as the heart has been in a great measure emptied, the blood, yielding to the reaction of the aorta and pulmonary artery, returns partly towards the heart, depresses the sigmoid valves, so as to form a solid floor, which supports not only the retractile action of the arteries, but also all the weight of the column of blood which they contain; at the same time that the blood, which penetrates into the cavity of the ventricle through the mitral and tricuspid valves, which are depressed under the influence of causes, which have been mentioned, is thrown against the arterial valves, to furnish them a solid point of support. The ventricle is filled with blood, and then recommences the series of actions which we have just described.

What is the principle of the movement of the heart? We are very far from the age when these movements were explained, by the presence of a concentrated fire (Galen), by an explosion as sudden as that of gunpowder (Descartes), or by animal spirits, the Archeus (of Van Helmont).

Without wishing to inquire what is the first cause of these movements, a research always as difficult as unprofitable, it is, however, interesting to know whether, like the other muscles, the central organ of the circulation is subject to the influence of the nervous system. But as the nerves belonging to the heart arise from two sources, the cerebro-spinal system and the ganglionic, it is necessary to know, what are those which give to the heart the principle of its movement. It is in vain, that Le Gallois has sought to place the cause of the motions of the heart in the nerves which spring from the spinal marrow; this theory, already combated by Bichat, before even it was presented by Le Gallois, has been overthrown by the

facts adduced by Welschius, Ruysch, Lallemand, and others, all of whom establish the fact, that in children deprived of the spinal marrow the heart executed movements; this theory is also annulled by the experiments of Wilson Philip, Mayo, Clift, &c., who have seen the action of the heart continue after the destruction of the spinal marrow, and even after the complete detachment of the organ of the circulation. Experiments are no more favourable towards the influence of the eighth pair; it was then rational to conclude, that the vital cause of the movements of the heart was in the ganglionic system, but it was difficult to confirm by actual experiment such an assertion; M. Magendie, whose skill is known, has never been able to succeed in these experiments.

M. Brachet, being more fortunate, saw the pulsations of the heart suddenly cease, in consequence of the section of the cardiac plexus or ganglion. However little confidence we may place in the results of an experiment, so laborious and so difficult to perform, we think, from what has been previously said, that the great sympathetic furnishes the principal motor power to the heart; the more so, since, like all the muscles supplied by the organic or ganglionic system, this organ is entirely beyond the domain of the will; whence it follows, that all pretended stories concerning persons who could, by will, suspend the movements of the heart, must be regarded as so many fables in physiology. It only remains for us to notice a very singular opinion of M. Mayo, upon the primitive cause of the movements of the heart. This physiologist thinks, that the motor principle may be found in an inherent power, totally independent of the nervous system, and which belongs to involuntary muscles.

It is in virtue of this power, that the heart contracts and dilates alternately for a certain time, in the absence of an external impression; in very active animals, such as the turtle-dove, he has seen the alternate movements of the heart continue for a very long time (an hour) after the heart has been taken from the chest. It is not our intention to enter into this discussion; whatever may be the moving power, whether it reside in the nervous system or is innate, is a matter of little consequence. It only concerns us to know, that the central organ of the circulation is subjected to a stimulus — the blood — which, arriving at definite intervals, and in proper quantity, is sufficient to maintain the regularity of its pulsations.

SECTION SECOND.

OF THE SOUNDS OF THE HEART.

If the ear, either naked or aided by the stethoscope, be placed over the præcordial region of a healthy person, two successive sounds

are distinctly heard, followed by an interval of silence or repose.* Of these two sounds, the first is more dull and prolonged than the other, and is very similar to the sound that is produced by drawing on a thick cord in a state of tension, and is perfectly synchronous with the shock of the apex of the heart against the thoracic parietes, or the impulse, and with the pulse in the arteries near the trunk (for, in the arteries which are far from the trunk, the radial and pedal artery, for example, there is always between the impulse of the heart and the arterial diastole a very short, but still an appreciable interval).

The second sound, which is shorter, clearer, and more sonorous than the first, has been compared by Laennec to the flapping of a valve, or the noise made by the lapping of a dog. Too much value must not be attached to these comparisons; the sounds of the heart are peculiar to themselves, and, in order to be well acquainted with them, it requires a great deal of exercise in the art of auscultation. The second sound succeeds the first very quickly, so that there appears at first to be no interval: but when you auscult persons with whom the pulse is very slow, at forty pulsations, for example, the interval which separate these two sounds becomes very easy to detect. It is this interval which M. Gendrin has proposed to call *peri-systole.* As has been mentioned before, the sounds of the heart are followed by an interval of silence or repose; this interval, much more considerable than the preceding, augments, like it, when the pulsations of the heart are slow; this M. Gendrin has proposed to call *peri-diastole.*

The sounds of the right side of the heart and those of the left side, take place at so short a period of time from each other, that it is very difficult to distinguish them in health. Laennec thinks that the sounds heard at the inferior portion of the sternum belong to the right cavities, and that those of the left cavities are heard between the cartilages of the ribs; he says, however, that the sounds are similar and equal on both sides.

However much respect we may entertain for the illustrious author of auscultation, we still must say, in accordance with the opinions of Hope and Bouillaud, that the sounds which are perceived under the inferior border of the sternum, and which belong to the right cavities, are much clearer than those which are heard to the left of the nipple, under the cartilages of the fifth and sixth ribs, and which belong to the left cavities.

At what point are the sounds of the heart at their maximum? it is at the point where there is found dulness in the præcordial region, about an inch above the apex of the heart, that the maximum of intensity of the first sound is heard, to which M. Pigeaux has given the name of *inferior sound.* On the contrary, the second sound, when at its maximum, is on a level with the arterial orifices, or

* These sounds, with which Galen was perhaps acquainted, and which Harvey pointed out, have been only perfectly studied since the time of Laennec.

with the inferior border of the third left rib near the sternum, and thence ascends up the course respectively of the aorta and the pulmonary artery, for the extent of two inches. The same author has given this the name of *superior sound.*

Concerning the intensity of the sounds of the heart, and the extent over which they are perceptible, we refer to what will be said under the articles *Dilatation* and *Hypertrophy.*

The existence of these two sounds has excited a great deal of interest among physiologists; explanations and theories have never been wanting, some very ingenious, the greater number neither creditable to the understanding nor the reputation of their authors. We intend to enumerate these different theories, all of which may be arranged in five groups; the *first group* will comprehend all the theories in which the two sounds are explained by the contraction and the dilatation of the cavities of the heart; the *second*, those which refer the sounds of the heart to its impulse against the parietes of the chest; the *third*, those which attribute these sounds to the friction of the blood; the *fourth*, those in which these sounds are explained by the play of the valves; the *fifth*, and last, will contain the mixed theories.

A. *Theories of the Muscular Sound.*

The most natural idea which would occur to persons who heard the sounds of the heart, for the first time, would be, that they were the result of the contraction of the different cavities of the heart. This theory, partially known to Galen, Harvey, Senac, and Haller, has been for the first time formally set forth by Laennec. "Of the sounds of the heart, the one clear, sudden, analogous to the flapping of a bellows valve (*second*, or *diastolic sound*), corresponds to the systole of the auricles; the other, more dull and prolonged (*first*, or *systolic sound*), coincides with the beating of the pulse, as well as with the sensation of the shock which announces the contraction of the ventricles." As to the manner in which these two sounds are produced, Laennec says nothing: he does not enter any further into the explanation of this phenomenon. M. Marc D'Espine, taking up more recently the theory of Laennec, alleges, that the first sound depends on the contraction of the ventricles, and that the second sound is caused much less by the contraction of the auricles, than by the dilatation of the ventricles.

B. *Theory of the Impulse of the Heart against the Parietes of the Chest.*

According to M. Magendie, to whom this theory belongs, the two sounds of the heart depend on a double impulse against the walls of the thorax; the first, or the *dull sound*, results from the impulse of the apex of the heart against the interval which separates the

sixth and seventh ribs, at the moment of its contraction; whilst the second, or the clear sound, is produced by the impulse of the anterior part of the heart against the sternum, at the moment of its dilatation.

C. *Theory respecting the Sound of the Friction of the Blood.*

According to M. Pigeaux, the first sound (*inferior sound*), coinciding with the contraction of the ventricles, is produced by the friction of the blood against the walls of the ventricles, the orifices, and the parietes of the large vessels: and the second sound (*superior sound*), coinciding with the contraction of the auricles, results from the collision of the blood against the parietes of the auricles, the auriculo-ventricular orifices, and the cavity of the ventricles. Hope had proposed, in 1831, an analogous theory, which he has since abandoned. Admitting, with the majority of physiologists, that the first sound was due to the systole, and the second to the diastole of the ventricles, he established at this period the fact, that the first sound is the result of regular vibrations in the mass of blood which fills the ventricles, by the currents and counter-currents produced when the blood is driven by the contraction of their walls through the arterial orifices; whilst the second depends on the reaction which takes place in the liquid which fills the cavity of the ventricles at the moment when the dilatation of the latter stops abruptly or suddenly. This theory has been espoused lately, in a somewhat modified form, by M. Gendrin, who has ably sustained it. According to this author, the vibrations which result from the sudden change of form which the blood experiences during the contraction of the ventricles, accounts sufficiently for the first sound (*systolic*); whilst the second sound (*diastolic*), is explained by the percussion caused by the column of blood, coming from the auricles, on the base of the ventricles, and upon the sides of the closed sigmoid valves.

D. *Valvular Theories.*

The valvular theory is one of those which have the most adherents: it was advanced by Drs. Billing and Bryan in England, and Rouanet in France, almost at the same time; but it appears, from a fact mentioned in the thesis of M. Marc D'Espine, that the celebrated professor of pathological anatomy, Dr. Carswell, had conceived the idea of it in 1831. Whichever may be the case, this theory owes its success chiefly to the diligent labour and persevering efforts of M. Rouanet. In the opinion of this cautious observer, the first sound is caused by the sudden tension of the mitral and tricuspid valves during the ventricular systole; the second sound results from the straightening or closure of the semilunar valves

of the aorta and pulmonary artery, produced by the reaction of these arterial tubes, and the rebound of the column of blood. In adopting M. Rouanet's theory, M. Bouillaud has thought proper to modify it; thus, he places, among the elements of the first sound, the sudden recoil of the semilunar valves against the sides of the aorta and pulmonary artery; and amongst those of the second sound, the sudden collapse of the auriculo-ventricular valves.

E. *Mixed Theories.*

Dr. Turner, who first sought to overthrow the theory of Laennec, referred, like him, the first sound to the contraction of the ventricles; but he thought, without affirming it in an absolute manner, that the second sound was due to the falling of the heart against the pericardium, at the moment of the ventricular diastole.

Dr. Corrigan thought that the first sound and the impulse were owing to the entrance of the blood into the ventricles, under the influence of the contraction of the auricles; as regarded the second sound, it resulted from the collision of the internal surfaces of the ventricles against each other, at the end of the systole of these same ventricles.

In the theory of M. Beau, the first sound depends on the impulse of the apex of the heart against the fifth intercostal space (as in M. Magendie's theory); it corresponds to the *auricular systole*, and to the *ventricular diastole!* The second sound corresponds to the muscular rising which is accomplished in the second or third intercostal space, and results from the *sudden dilatation of the auricle!*

Dr. Williams, attributing both sounds to the systole and its effects, regards the first sound as the direct result of the impulse of the apex of the heart against the thoracic parietes, at the moment of its systole (as in the theory of M. Magendie); and the second sound, as being the result of sonorous vibrations impinging on the mitral and tricuspid valves by the blood which the ventricles force before them.

In the opinion of Mr. Carlile, the first sound is produced by the projection of blood out of the ventricles, (as in the theory of M. Marc D'Espine,) and the second, by the obstacle which the semilunar valves oppose to the return of blood into the ventricles.

It remains to introduce the last theory, which we adopt with some modifications, and which appears to us to meet all objections, whilst at the same time it is consecrated by that supreme test, experience; this theory is the one which has been advocated by Hope in the last edition of his *Treatise on the Diseases of the Heart.* We regret that the brief space allotted to the "Manual" will not permit us to introduce here the curious experiments by which the author supports his theory; we shall have occasion to cite some of them in another place, when we examine all the preceding theories separately. The following is the theory of Dr. Hope:

In his opinion, the *first sound* is composed — 1st, of the extension of the auriculo-ventricular valves; 2d, of the sound of muscular extension, a loud and smart sound resulting from the movement of sudden extension which the contracted walls of the heart experience at the moment when the auriculo-ventricular valves are closed; 3d, of the *muscular sound*, a dull sound which accompanies commonly the contraction of all muscles, and the nature of which we shall soon investigate. The valvular extension gives the sharp and strong character to the commencement of the first sound; the sound of muscular extension adds still more force to the valvular sound, but it renders it at the same time more dull; the muscular sound prolongs gradually the first sound until the end of the act of contraction. The *second sound* results from the sudden expansion of the aortic and pulmonary semilunar valves, produced by the return stroke of the column of blood.

Before furnishing the proofs which tend to establish this theory, let us cast a glance at those which precede it.

A gentleman, who joins to soundness of mind extensive knowledge, M. Littré, well detected the difficulty, when he said: One constant point of departure which depends on no theory, "is that the first sound is produced in the systole of the ventricles, and the second in their diastole. Every explanation which should deviate from this line, would be false on that account alone." In this way may be disposed of, without need of discussion, the theory of Laennec, which refers the clear sound, or second sound, to the auricular systole, and the theory of Corrigan, which refers the first sound to the impulse of blood which, coming from the auricles, strikes against the ventricular parietes; as also the theory of Williams, which attributes the second sound of the heart to the systole of the ventricles and to their effect; and finally, the theory of Beau, which makes the first sound coincide with the diastole of the ventricles, and the second sound with the diastole of the auricle.

The muscular theory has for its basis the existence, under certain circumstances, of a peculiar sound, which is called muscular or rotatory sound; the researches of Erman, of Wollaston, and of Laennec, have proved, that when the ear is applied over a muscle which is contracted, there is heard a noise very similar to that of a carriage moving rapidly at a distance: this sound is composed of many successive sounds, approaching nearer to each other in proportion to the increase of the muscular contraction. One can easily be convinced, by applying the ear over his wrist strongly contracted, that there is no relation between the sound called *rumbling* or *muscular*, and the normal sounds of the heart; the first are composed of successive intervals, the others of two short and clear sounds separated by a very appreciable interval. Moreover, in this theory, it is impossible to explain the second sound, since this sound coincides with the diastole or the relaxation of the ventricles, and the muscular sound is only produced during the contraction

of the muscles. This theory, therefore, does not deserve to be retained.

Does the theory which refers the two sounds of the heart to the impulse of this organ against the thoracic parietes, deserve any further confidence? Certainly not. Pathological facts prove conclusively, that the presence of liquid or membranous products interposed between the heart and the sides of the chest, rendering almost impracticable the impulse of this organ, does not prevent the two sounds from being heard; lastly, the experiments of Hope and Bouillaud have annihilated this hypothesis, by demonstrating that the sounds of the heart continue after the removal of the anterior part of the thorax.

Can it be true, that the impulse and the friction of the blood against the parietes of the ventricles and of the large arterial trunks, will account for the double sound of the heart? Let us first inquire respecting the nature of the sounds which result from the friction of the blood against the smooth, polished parietes of the arterial trunks and of the heart. The physical experiments made on perfectly smooth tubes, and on the dead subject, prove that in all circumstances there is produced a bellows sound having nothing analogous to the flapping which characterises the sound of the heart.

We do not deny, that the entrance of blood into the cavities, either ventricular or arterial, produces a sound; but it is our opinion, that this sound is merged entirely in the normal sound of the heart. Therefore, the sounds of the heart cannot be explained by the friction of the sanguineous column against the parietes of this organ and of the arteries. Can we explain them by the collision of the molecules of the blood, as Hope, and also Gendrin, have done?

M. Gendrin's opinion is, that the systolic sound corresponds to a systolic contraction of the heart, in consequence of which this organ changes its form, and tends to become spherical; the liquid molecules, pressed by the contraction of the ventricles, advance towards the orifices at the base; but they meet, in the closure of the auriculo-ventricular valves, an obstacle to their return into the auricles; and being pressed against each other, they produce vibrations, which are transmitted to the contracted walls of the heart, and thence to the solid parts. Such is the account of the first sound.

If the first sound was owing to the cause assigned by M. Gendrin, it would not have its maximum at the apex of the heart; for it is not there, but rather towards the arterial orifices, that these sanguineous molecules converge; and consequently the strongest collision takes place where the most considerable vibrations occur. In rare cases, where the two auriculo-ventricular orifices and their valves are diseased at the same time, the first sound disappears entirely, and is replaced by a bellows sound: another proof, that the first sound is not due to the change in the form of the column of blood. In regard to the diastolic sound, M. Gendrin thinks that

the blood, entering into the ventricles, fills them instantaneously, with all the rapidity which a liquid undergoes when obeying a *vis à tergo* force, and strikes quickly against the parietes of the ventricles of the heart, especially towards the superior part of the cavity, because it is on this part that the force and rapidity of the impulse of a liquid which penetrates into the ventricles are spent. It is, observes M. Gendrin, a phenomenon, analogous to that which is seen in the experiment of the water-hammer, or still more, to the mercurial column in a barometer when put in motion. We are again forced to oppose this explanation. There is no proof, either from pathological facts, or experiments, that the blood, in entering into the ventricles, undergoes, at the apex of the heart, a movement of reflexion, in consequence of which it would be carried towards the base; even admitting this, neither the experiment of the water-hammer, nor that of the barometrical chamber, can support this opinion, since these two phenomena take place in a vacuum, and it has been shown that the ventricles never empty themselves entirely of the liquid which they contain.

At the time when M. Bouillaud published the first edition of his *Treatise on the Diseases of the Heart*, he thought that the direct or experimental proofs of the valvular theory failed completely, and that, perhaps, they always would fail. But this has not been the case; the beautiful experiments of Dr. Hope have taught that, as often the auriculo-ventricular valves are prevented from closing the corresponding orifice, the first sound diminishes considerably in intensity without disappearing entirely. (It is on this experiment that Hope relies for the purpose of admitting the existence of the rotatory sound, and of the sound of muscular extension.) If the first sound is only partially produced by the extension of the mitral and tricuspid valves, the second sound is produced entirely by the extension of the semilunar valves. Hope has proved this fact, by showing — 1st. That the compression of the aorta above the arterial orifices, so as to prevent the return of blood to the semilunar valves, caused the second sound always to cease. 2d. That if you prevent the valves of an artery from closing the orifice, the second sound is diminished, and, if the same experiment is tried in the larger arteries, the second sound disappears completely, and is replaced by a murmur from regurgitation. It is necessary to give the reasons upon which Hope has thought proper to place, among the causes of the first sound, *the muscular sound and the sound of muscular extension.*

This excellent observer has proved, by his experiments, that when the left ventricle is opened, and the finger introduced, so as to efface for a moment the cavity of the right ventricle, and consequently when it is placed in the circumstances when the valvular sounds and the sound of muscular extension should cease, there still is heard the first sound, but *dull* and *obscure*, and very similar to the muscular sound, which is artificially produced. This muscular sound probably, in the opinion of Dr. Hope, increases the intensity of the first

sound, and giving it a dull character, it contributes, likewise, to prolong the first sound. The proof, that this prolongation does not belong to the flapping of the auriculo-ventricular valves, is, that it is wanting under certain circumstances, and then the first sound approaches nearer the second. Thus it happens, that when the heart is enfeebled, whether by a disease belonging to itself, or depending on a general cause, this prolongation, or the muscular sound, is almost completely deficient.

These are the words of Dr. Hope: "At each systole of the ventricles, their sudden tension was such as to produce a sudden shock, which raises the finger. The first sound coincides exactly with the shock. This impulse by lateral expansion is greatest at the margins of the auriculo-ventricular orifices; it is at this place that the finger is thrown out with a violent jerk." Such are the facts upon which the author relies, in order to admit the existence of a sound of muscular extension. "The first sound," says he, "preserves its character as long as the sudden shock of muscular extension is present, but as soon as it disappears, the first sound becomes dull and obscure."

The reasons given by Hope do not appear to us to be entirely conclusive; he perfectly understood it himself, when he adds: "It may be objected that the sound was occasioned not by muscular but by valvular extension, and this argument is fair."

We do not, then, admit the sound of muscular extension; we admit that the extension of the auriculo-ventricular valves is accompanied by an appreciable noise; but does it necessarily follow that this sound, in connexion with the muscular sound, are the only elements of the first sound? We cannot think so; but there seems to have been no notice taken of a very important cause; we allude to the shock of the column of blood against the sides of the auriculo-ventricular valves; these valves vibrate, not only because they are submitted to a violent extension, but because they are struck by the wave of blood. It is of little importance, however, whether the valves are set in vibration by the extension or the shock of the liquid, since the result is the same. What we admit for the first sound, we admit also for the second, and think that the return stroke of the blood against the floor of the closed semilunar valves, is one of the principal elements of the second sound.

To resume: our belief is, that the first sound is composed, first, of the sudden extension of the auriculo-ventricular valves; second, of the shock of the column of blood against the sides of the valves; and third, of the muscular sound; the second sound is owing, first, to the sudden extension of the semilunar valves, and second, to the rebound of the column of blood against the sides of these same valves.

As to the sounds which are produced by the impulse of the apex of the heart against the thoracic parietes, or by the friction of the column of blood against the walls of the ventricles, the orifices and large vessels, we do not deny their existence, but we think that they are entirely concealed by the normal sounds of the heart. Here-

after, when we study the pathology of the heart, we shall see that these sounds gain force, are distinct from the normal sounds, equal them, and sometimes efface them entirely, under the influence of pathological conditions of this organ.

CHAPTER III.

PATHOLOGICAL PHYSIOLOGY OF THE BEATS AND SOUNDS OF THE HEART.

SECTION FIRST.

PATHOLOGICAL MODIFICATIONS OF THE BEATS OF THE HEART.

We shall first direct our attention to the beats or pulsations of the heart, independently of the accompanying sounds.

In the healthy state, the heart strikes by its point between the cartilages of the fifth and sixth left ribs, at four or five centimetres, or more than an inch, from the left border of the sternum. When the hand is placed over the præcordial region, in a healthy man, of moderate fulness of frame, and whose heart is well proportioned, there is experienced the sensation of a body, which corresponds to a small extent of the sides of the chest, and which communicates only a very feeble shock to the adjacent parts. In very fat persons, the space in which it may be detected is much more limited, and often it is very difficult to recognise the apex of the heart: in lean persons, with narrow chests, especially in young children, the beating of the heart may be perceived in all the left anterior portion of the chest, sometimes even in all of the right side. The pulsations of the heart in the healthy state coincide with the systole of the ventricles, and the impulse which is perceived in the præcordial region, is almost exclusively due to the left ventricle, which forms of itself nearly all the apex of the heart.

This being settled, let us inquire what are the modifications which the pulsations of the heart experience — 1st, in their seat; 2d, in their force; 3d, in their extent; 4th, in their rhythm.

1st. *Modifications in the Seat of the Heart's Pulsations.*

Most of the diseases which induce changes in the form, volume and position of the heart, cause, consequently, a displacement of the apex of the heart; sometimes, and indeed most often, the apex alone

changes its position; sometimes it only follows the movement which has been impressed on the base of the organ. It is always easy to recognise, by the place where the systolic impulse of the heart is perceived, whether the apex is depressed or elevated; but it is less easy to find out whether the base is also displaced at the same time. Still, it is possible to attain this knowledge, by observing where the second or diastolic sound is seen at its maximum, as well as the character of the impulse. When the base of the heart is depressed, the maximum of the second sound is much below the synchondro-sternal articulation of the third rib; and the anomalous impulse accompanying the diastole is equally felt much below the third rib; if, to these characteristics, be joined the depression of the apex of the heart, there can be little doubt respecting the depression of the whole organ. These displacements, which are always very rare, may take place *directly downwards* (as in the case where tumours press on the base of this organ); *directly upwards* (for example, when the diaphragm is strongly forced back towards the chest); *laterally* (by tumours situated on the lateral parts of this organ, by pleuritic effusions, or in the case of a general transposition of the viscera); *backwards* (by tumours of the mediastinum, and aneurisms of the aorta); finally, *in different ways* (by adhesions of the pericardium, distortions of the spine, &c.).

Most commonly, displacements of the whole heart are the result of alterations in neighbouring parts; but this is not the case with displacements of the apex, which are always owing to an augmentation of volume, or alterations in the form of this organ, and which take place generally in two ways; downwards and outwards, or downwards and inwards.

In the last case, which most rarely happens, the apex of the heart approaches nearer to the xyphoid cartilage, and the impulse only raises the chest to a very limited extent. These displacements ordinarily are caused by a well-marked hypertrophy of the base of the left ventricle, with distension of the corresponding auricle. The displacements downwards and outwards, in which the apex of the heart descends below the sixth rib, and is ten or twelve centimetres, or about four inches distant from the left border of the xiphoid appendix, and in which the impulse increases in force and duration, are the most common, and depend especially on diseases of the left ventricle (hypertrophy with dilatation), conjoined with distension of the right auricle.

2d. *Modifications in the Force of the Pulsations of the Heart.*

The force of the heart's pulsations varies to an almost infinite degree; being sometimes so feeble, that it is very difficult to recognise them, at other times extremely violent. The modifications of force may be either transient or permanent; thus, the action of the heart is seen to increase under the influence of fever, of a moral

emotion, of fatiguing exercise, &c., and to resume its normal character after the cessation of these causes; the action of the heart is also seen to diminish under the influence of certain medicinal agents, as digitalis, or in certain diseases of the nervous centres. The permanent modifications of force alone merit our attention; in these, the force of the heart's pulsations is in a direct ratio with the thickness of its parietes; everything else being equal, the more thickness the parietes possess, the more vigorous will be the impulse. Thus, in hypertrophy, the impulse is sufficiently strong to raise the head of the observer, sometimes even to produce a disagreeable shock to the ear: whilst, in dilatation, the impulse is scarcely sensible, and when it is so, it is only a sudden and short impulse, which causes the thoracic parietes to vibrate, without having sufficient power or sufficient duration to raise them. It is thus, that if a muscle which is thin has less power, it has, in compensation, more facility and rapidity in its movements than a thick muscle; in other words, if the apex of the heart is carried more rapidly forwards, it seems, as it were, to exhaust its energy in this act. It is not so in the disease called hypertrophy with dilatation; the impulse borrows from the hypertrophy all its force, and preserves the abrupt and dry character of dilatation.

The strength of the pulsations of the heart may be modified by causes situated external to the organ; hence, whatever separates the heart from the sides of the chest (liquid effusions into the pericardium and adipose enlargements of the heart), enfeebles the impulse; whilst this latter is seen to increase under the influence of causes which force the heart nearer to the sides of the chest (aneurisms of the aorta situated behind the heart, and adhesions of the pericardium).

3d. *Modifications in the Extent of the Pulsations of the Heart.*

Their extent, like the force of the pulsations, is susceptible of great variation. The general rule is, that it is directly proportionate to the strength, or rather to the thickness of the walls of the heart. If it is increased in thickness, the pulsations raise the chest to a great extent; often even the whole sternum and all the left side of the thorax are affected at each systole of the heart, with a movement of elevation, which is very appreciable to the naked eye. If it is diminished in thickness, or has lost its consistence, the pulsations of the heart lose so much in their extent, that often it is very difficult to find them. Like those of force, the modifications in extent in the pulsations are either transient or permanent; and, in the last case, they may depend either on a lesion of the heart and its appendages, or on a lesion of the neighbouring organs.

4th. *Modifications in the Rhythm of the Pulsations of the Heart.*

The modifications of the rhythm may take place as respects *number*, *duration*, and *order of succession*, of the pulsations of the heart. 1. In relation to *number:* In a variety of morbid states of the heart, whether idiopathic or sympathetic, the pulsations are observed to amount to a hundred and forty or one hundred and sixty *per minute*, or fall to thirty, and even to seventeen. This modification in frequency is not the only one, which the movements of the heart experience, in reference to their number. In the healthy state, to each revolution of the heart, formerly called each systole of the heart, there is a corresponding impulse (*systolic impulse*); but it is not thus in certain morbid states; in hypertrophy, and especially in hypertrophy with dilatation, the systole and diastole are both accompanied by an impulse. This *diastolic impulse*, which had been discovered by Laennec, is a valuable sign in the diagnosis of a large hypertrophy, and especially of one complicated with dilatation of the heart. 2. *Duration:* In a healthy man, the pulsations of the heart consist in a slightly marked and rapid elevation. When the heart loses its thickness, this elevation takes place still more quickly, because the heart being thin, contracts with much more facility and more rapidly. But when the heart is increased in thickness, it is not at all the same; the hypertrophied ventricle, difficult to move, contracts slowly and gradually; it seems, says Laennec, that the heart, swollen out, applies itself to the sides of the chest, at first by a single point, then by all its surface, and then falls back to its proper dimension. As to, 3, the *order of succession:* Sometimes, after a certain number of beats, the heart stops and rests during an interval equal to an ordinary pulsation; these are called *intermissions*, and are distinguished into *true and false intermissions*, according as they are owing to a real suspension of the action of the heart (*arrest* or *hesitation of the heart*), or to a too feeble contraction of this organ. They may present themselves in a regular manner after a certain number of pulsations. Sometimes the pulsations succeed each other at unequal intervals, the force of the impulse remaining the same, or varying at each instant. These are called *irregularities;* and here, one of two things must take place: either the rhythm is completely deranged, and the pulsations which succeed bear no resemblance to each other; or these pulsations are similar, which gives them, with certain restrictions, their regular character. It is difficult to form an idea of the disorder which reigns in the actions of the heart during these irregularities; the systoles and the diastoles succeeding each other in the most irregular manner. According to some, it is possible to count, in a single contraction of the ventricles, two and three diastoles of these cavities, and *vice versâ*. When we speak of the sounds of the heart, the opinions on this point will be examined.

SECTION SECOND.

PATHOLOGICAL MODIFICATIONS OF THE SOUNDS OF THE HEART.

The sounds of the heart may be modified, 1st, by their *seat;* 2d, by their *force;* 3d, by their *extent;* 4th, by their *rhythm;* 5th, by their *tone;* 6th, and last, they may be *concealed* and *replaced* by anomalous sounds.

1st. *Modification in the Seat of the Sounds of the Heart.*

These modifications bear a close relation with the modifications in the position of this organ. When the heart is entirely displaced, its two sounds are displaced also; but where there is less complication, there exists always between the maximum point of the two sounds the same interval as in the healthy state. When, on the contrary, the apex alone is displaced, (which is the most frequent accident,) there is only one sound of displacement; it is the systolic sound, whose maximum is much further from, or much nearer than usual to, the maximum point, where the diastolic sound is perceived. It is well understood how these displacements may take place in all ways; upwards, downwards, backwards, and laterally, according to the position in which the heart is placed.

2d. *Modifications in the Force of the Sounds of the Heart.*

These sounds may increase or diminish in intensity, which may depend on the state of the neighbouring organs, or of the heart itself. It is thus that the sounds of the heart are perceived to grow feebler by some cause (such as emphysema, effusion into the pericardium, &c.), where the heart is separated from the sides of the chest, and covered by bodies which are bad conductors of sound, whilst they are seen to augment in force under opposite circumstances. As to the augmentation of the sound, which depends on the heart itself, it takes place under the influence of causes that act on the *dynamic* powers of this organ, sometimes under the influence of material modifications. In this last case, and it may be regarded as a general rule, the strength of the sounds is in an inverse ratio to the thickness of the walls of the ventricles. In simple hypertrophy, the first sound becomes more dull in proportion as the hypertrophy is more considerable; it is almost extinguished when the hypertrophy becomes extreme. The second sound is equally enfeebled, and this is easily understood, since the contraction of the ventricles takes place slowly, and the extension of the valves also is slower than common. In dilatation, the first sound

becomes strong and clear, like the second; the second sound increases more or less in force, because the muscular contraction takes place with great rapidity, and there is rapid tension of the valves. But it is especially in hypertrophy with dilatation that these two sounds reach their maximum; for, in this disease, the heart adds to great rapidity of action considerable force and vigour; in consequence of which, there is great rapidity and violence in the extension of the valves, and also excessively loud sounds.

3d. *Modifications in the Extent of the Sounds of the Heart.*

These modifications are generally in a direct proportion to the force of the sounds of the heart, and in an inverse ratio to the thickness of its walls. Besides, their extent varies widely, according to the thickness of the organ. Thus, whilst in simple hypertrophy these sounds are limited to a very small space, in simple dilatation, and especially in dilatation with attenuation, they are perceptible on all the left side, sometimes even over the whole right side of the chest.

4th. *Modifications in the Rhythm of the Sounds.*

These modifications may, and most often do, bear a relation to the *number* of pulsations; but, sometimes, instead of two sounds for each revolution of the heart, three, and even four may be heard. In other cases, only one sound is perceived for each revolution; or there may be no sound perceived at all. This happens in the last stages of the diseases of the heart. When there is only one sound extinguished, it is ordinarily the diastolic sound. When, on the contrary, these sounds amount to three or four, it generally depends on the contraction of the left auriculo-ventricular orifice, and these numerous sounds appear to affect the diastolic sound; they depend, in the opinion of most authors, on the diastolic sound not being accomplished at the same time in the left and right cavities of the heart. Hope, on the contrary, regarded these sounds as badly observed, and thinks that these triple and quadruple sounds are the result of the irregular pulsations of the heart, which is too feeble to transmit the impulse to the radial artery, but that this impulse is always perceived in arteries near the trunk.

Whichever opinion be sanctioned (and we do not adopt that of Hope), the fact remains, and it is important, since it is sufficient of itself, when permanent, to indicate, in the majority of cases, a considerable contraction of the left auriculo-ventricular orifice. In relation to *their duration;* in hypertrophy, the first sound is prolonged, like the *sound of a file*, when the column of blood is about to clear a contracted orifice. As regards the *order of succession*,

there are modifications; as, for example, when the sounds succeed each other, at unequal intervals, which most always takes place in palpitations.

5th. *Modifications in the Tone of the Sounds.*

The tone of the sounds varies widely, sometimes more clear or more dull than in the normal state; bearing a general relation to the thickness of the parietes: at other times, it is hard and dry, like parchment, or harsh and rough, or hoarse. These modifications are dependent most frequently on an increase or diminution in the rigidity of the membranous folds of the valves.

6th. *Modification of the Sounds of the Heart by Anomalous Sounds.*

These anomalous sounds are of two kinds; some have their origin in a material cause, whose nature and effects are perfectly known; the others cannot be referred to any apparent lesion of the solids. The first have received the name of *organic anomalous sounds;* the last are called *inorganic anomalous sounds.*

A. *Organic Anomalous Sounds.*

These sounds are of two kinds; some are produced external to the heart, some in the interior of this organ.

1st. *Intra-cardiac murmurs.* — Diseases of the valves, which are almost always the cause of these murmurs, impress on the sounds of the heart various pathological alterations, and transform them into sawing, bellows, filing, rasping, whistling, and even into true musical sounds.

It is well known that Laennec, in the first edition of his immortal work, after having referred all sounds of this nature to organic lesions, or rather to contractions of orifices, omitted in the second edition so explicit an assertion; and, puzzled by the great number of facts which were arranged under this head, he admits, in order to explain all these anomalous sounds, a spasm of the heart, a peculiar disorder of the nervous system. It is not our intention now to defend auscultation against its author; time has exposed the errors of Laennec. This author did not recognise the sound which has been since designated that of regurgitation, or the impossibility of the valves closing an orifice, and preventing the return of blood into the cavity from which it has just been expelled. Laennec did not consider the state of the blood itself. Without the discovery of the murmurs from regurgitation, which we owe to Elliotson and Hope, the pathology of the heart would be still obscure.

Before examining under what circumstances these murmurs are

met with, let us begin by establishing this fact, that the first sound, the contraction of the ventricles, the passage of the blood through the arterial orifices, the arterial diastole, are phenomena perfectly synchronous with each other, and that the second sound, the auricular contraction, the passage of blood into the ventricles, the diastole of these cavities, the systole of the arteries, are also synchronous acts.

As often as the column of blood, impelled by the ventricles, finds in the arterial orifices an obstacle to its passage, (arising from pathological alterations, inflammatory or otherwise, from fibrous, fibro-cartilaginous, steatomatous, or osseous degenerations, vegetations, sanguineous concretions,) which diminish its diameter, there will take place a murmur, coinciding with the first sound, the contraction of the ventricles, &c., &c., which is called *systolic murmur*, or *murmur from diminution of the arterial orifices.*

Whenever the valves of the arterial orifices, in consequence of alterations, (various degenerations of the base or summit of these valves, perforative atrophy, anomalous adhesions, polypous concretions,) become unable to close completely the arterial orifices, and to prevent the return of blood into the ventricle, a murmur will result, coinciding with the diastole of the ventricles and the second sound (*diastolic murmur*, or *murmur from regurgitation*).

If, from any cause whatever, the diameter of one of the auriculo-ventricular valves becomes diminished to any great degree, the blood, impelled by the contraction of the auricle, whilst passing through the contracted orifice, will cause a murmur corresponding to the diastole of the ventricles (*diastolic murmur*, or *murmur from diminution of the auriculo-ventricular orifices*).

Finally, if one of the auriculo-ventricular orifices does not act sufficiently well, to prevent the ingress of blood into the auricles at the moment of the contraction of the ventricles, there will be, from the reflux of the column of blood, a murmur synchronous with the systole of the ventricles (*systolic murmur*, or *murmur from regurgitation, by the auriculo-ventricular orifices*).

The diagnosis of valvular murmurs, and of the lesions which they indicate, rests entirely upon the order in the succession of the contractions of the heart's different cavities. Each orifice may be the seat of two murmurs, one of contraction, the other of regurgitation; a murmur abstractedly considered, may consequently belong either to a contraction or to a regurgitation. (It may be well to remember that the murmurs of the right cavities are very rare.)

M. Gendrin, bringing to the examination of the murmurs of the heart that analytical spirit which never forsakes him, has thought proper to establish still more numerous divisions; attaching to each one of the subdivisions of the heart's actions particular murmurs, he has admitted, among the systolic ones — 1st. *Præsystolic murmurs*, or those which coincide with the heart's contractions, and which indicate rugosities on the ventricular floor of the mitral and tricuspid valves. 2. *Systolic* murmurs, indicating obstacles to the

course of the blood, and the existence in the ventricular cavity of an insufficiency of the auriculo-ventricular valves, or a partial aneurism of the heart. 3d. *Peri-systolic* murmurs, or those which immediately follow the systole of the heart, and which point out obstacles seated at the origin of the large arterial trunks, or even in these vessels themselves. Among the *diastolic* murmurs, M. G. ranks, — 1st, *pre-diastolic* murmurs, indicating rugosities at the auriculo-ventricular orifices, or on the auricular floor of these valves; 2d, *diastolic* murmurs, showing alterations of the free extremity of these same valves; 3d *peri-diastolic* murmurs, proving the existence of contraction of the auriculo-ventricular orifice.

We have convinced ourselves of the facility with which M. Gendrin makes use of these subdivisions in diagnosis, but we do not adopt his plan, since we are convinced that it requires for its exercise a perfection or acuteness of sense which every one does not possess, and also, because these distinctions are not always practicable.

The valvular murmurs result from the collision of the sanguineous molecules among themselves, and against the solids which enclose them, when the liquid, in passing through the orifice of a cavity, is subjected to anomalous commotions. The sonorous vibrations which are obtained in these cases, are quite analogous to those which are seen when a liquid is made to circulate in a tube, and this liquid meets an obstacle; so that experience accords very well with the theoretical explanation which we have given of it. According to Corrigan, the production of the bellows sound is subordinate to the two following circumstances: 1st, the movement of the blood in the form of currents, (instead of an equal and natural movement,) tending to produce corresponding vibrations in the parietes of the cavities of the heart. 2d, a diminution in the tension of the walls of the arteries, or of the cavities themselves, which permits these walls to be easily set in vibration, by the irregular currents which are there produced. There is no doubt that the currents and counter-currents contribute to produce these murmurs: but how can they produce them but by inducing anomalous movements, and an increase of friction? The diminution in tension, which we admit in some special circumstances, is far from being a necessary condition for the production of a murmur: for, it is proved by experiment, that murmurs are produced in the tubes which are the thickest and most resisting, and that, consequently, the sides concur slightly, or not at all in the production of these murmurs.

Valvular murmurs present in their *tone* certain modifications, which Laennec has recognised under the name of bellows, sawing, filing, rasping sounds, continuous murmurs, whistling or musical sound. The bellows sound is a soft sound slightly raised, similar to that produced by an ordinary bellows; the *rasping*, *filing*, and *sawing* sounds evince more marked degrees of roughness.

Laennec thought, that the very harsh sounds (as filing, sawing,

rasping,) announced ossifications, whilst the very soft sounds indicated obstacles whose surface is smooth; consequently, fibrous or fibro-cartilaginous alterations.

But it is now proved, that if the nature of the obstacle has any agency in the production of these murmurs, or if ossifications, exposed and projecting into the cavity of the vessels, cause rough murmurs, there are often found, on the other hand, very extensive ossifications, especially when covered by the lining or internal membrane, which only occasion a soft murmur or a bellows sound; from which fact we must conclude, that the harshness of the murmurs is subordinate to the location of the obstacle, or of the contracted orifice, and requires that the current of blood be much broken, and that the liquid and neighbouring solids enter into considerable vibrations. Let us not, however, forget that the harshness of the sounds is dependent on a still more efficient cause, that is, the rapidity of the current of the blood.

Thus, a murmur having a harsh character (sawing sound, for example), is often known to descend by degrees to the filing and rasping sound, then to the bellows sound, under the influence of measures which diminish and render slower the action of the heart: and it has been known to disappear completely during the feeble contractions of this organ, and only to show itself during its violent ones.

It is because the current which traverses the arterial orifice is deficient in strength, that there is no harsh murmur in the case of deficient closure of the arterial orifices, or the contraction of the auriculo-ventricular orifices, and that sometimes in this last case, the murmurs are occasionally wanting.

These harsh murmurs give great accuracy to our diagnosis; they always point out an organic lesion; sometimes a considerable contraction, and rarely a deficiency; when they occur among persons advanced in age, they are probably caused by ossifications.

Continuous murmurs are very rarely observed in the heart, and the circumstances under which they occur are not well established. Hope, who has twice met with them, has seen it produced, in the first case, by the agitation of a small quantity of liquid which was churned between the folds of the pericardium, the latter being rendered rough by the presence of false membranes; and in the second case, by the reflux of blood from an aneurism of the aorta into the right ventricle. These continuous murmurs, like the sawing and rasping sounds, gain much more intensity, in proportion to the momentum of the column of blood which traverses the contracted orifice.

The musical sounds which Laennec had observed in the arteries, but never in the heart, are, however, very often met with in this last organ. They may consist of distinct notes; a whistling, cooing, and mewing sound, and sometimes the note is perfectly pure; but it is most commonly mixed with an ordinary bellows

sound; sometimes it precedes this murmur, sometimes it is more or less compounded with, and seems to make part of it; at other times, it takes place subsequently, and seems to be its *apogée*.

It is very seldom that the musical sound is supplanted by one of these sounds, which is capable of being distinguished. By the manner in which the musical sounds succeed the ordinary murmurs, it is evident that they are phenomena of the same kind, and are often confounded with each other. The musical murmur is owing to more numerous and more acute vibrations: but there is no more difference between these and the others, than there is between the blowing sound and that in common whistling by the lips. It is well known with what facility one passes into the other, it being only necessary, in order to insure this transition, to accelerate the exit of air without altering the position of the lips; it matters little whether the medium be liquid or aëriform. M. Cogniard Latour has proved, that the different degrees of strength, which are used to force a liquid through apertures, are sufficient to change a friction sound into a musical sound. The circumstances which produce musical sounds are not sufficiently well known; although Dr. Hope has thought himself justified in concluding, from his observations, that these sounds are accompanied rather by a deficient closure, than by a contraction, of the orifices of the heart.

The *elevation* or the *pitch* of the bellows, sawing, rasping, filing sounds, depends on the greater or less depth of the places whence these sounds proceed: the nearer these sounds are to the ear, the higher is the pitch, and reciprocally; everything else being equal, in proportion to the force with which the current traverses the contracted orifice, the louder are the sounds; in conclusion, the narrower the aperture, the more acute is the sound, provided, however, the current of blood be sufficiently strong.

It is at the orifice, or in the pulmonary artery, that the murmurs are most superficial, and, consequently, there they have a more elevated pitch. Most generally, the sound that they produce holds a medium between that caused by the repetition of the letter S, s s s s, and that which is produced by the repetition of the letter R, r r r r. For the same reason, the murmurs which are present in the ascending aorta, at the point where this artery approaches the sternum, have almost as high a key as that produced by the murmurs of the pulmonary artery.

The orifice of the aorta is a little more deeply seated; thus, the murmurs which take place at this orifice are rarely elevated above the sound produced by the repetition of the letter R This sound, according to Hope, is the true type of the sawing sound, although M. Bouillaud has represented it by the repetition of the letter S.

As the currents which occasion the diastolic murmurs, or those from regurgitation by the aorta and pulmonary arteries, are much more feeble than the currents which produce the systolic murmurs, the first murmurs are always lower. Hope, who regards them as

being two tones lower, has represented them by the sound which the prolonged aspiration of the English word *awe* (*â*) produces.

The auriculo-ventricular valves are still more deeply seated; therefore, the murmurs which occur near these orifices are still lower; Hope says, that they are four times lower, and are perfectly represented by the sound which the repetition of the vowels *oo* makes. Moreover, the key is a little higher, if there be a deficiency of the corresponding valves, on account of the force with which the current traverses the orifice: it is, on the contrary, at its minimum of elevation when there is contraction, in consequence of the feebleness of the column of blood which comes from the auricle. (The murmurs of the right auriculo-ventricular orifice are a little higher toned than those of the corresponding left one, because they are found at a less depth.)

The knowledge of the pitch or key of the murmur is not a thing of idle curiosity; it is only by this means that we can arrive at a correct diagnosis of a diseased valve; and this is practicable, because we are able to refer to the source of the murmurs, when we have once discovered their maximum. Let us add, that if these murmurs maintain their degree of elevation in proportion as they are more or less superficial, or rather as they are more or less reflected towards the centre of the chest, they also maintain this relation in whatever place we examine them; the sound which offers the character of the sawing sound in the præcordial region, when explored under the clavicle, may only consist in a dull humming sound.

Everything regarding valvular murmurs is so important, that we have thought proper to place here some general conclusions by Dr. Hope, on the nature and mechanism of the murmurs of this space. They do not present everything that could be desired; but they exhibit the exact state of the science up to this period.

1st. The *systolic* currents of the ventricles are stronger than the *diastolic* currents; consequently, the murmurs which are due to the first, are much more intense than those caused by the second.

2d. Rough murmurs, (as sawing, rasping, filing sounds,) may be the result of considerable contractions, salient and rough obstacles, whatever be their internal nature, but they can only be produced by the systolic currents, and never by the diastolic ones.

3d. The pitch of the murmurs becomes more elevated, in proportion to their proximity to the surface and to the strength of the current, and *vice versâ*. The key is lowered, independently of the depth, according to the distance of the murmurs.

4th. Musical murmurs indicate nothing more than the ordinary murmurs.

5th. Rough murmurs, and even the bellows sounds, when strong and permanent, indicate an organic lesion.

6th. Murmurs from regurgitation necessarily point out an organic lesion.

7th. Continuous murmurs, which are met with in the heart indicate, probably, sometimes an organic disease, accompanied with reflux of blood from the aorta into the right ventricle, or into the pulmonary artery, sometimes the agitation or churning of small quantity of serum, or other liquid between the folds of the pericardium, rendered rough by false membranes.

The study of the elevation or pitch of the valvular murmurs naturally leads us to investigate what are the points where these different sounds are at their maximum. This is undoubtedly the true basis of all differential diagnosis in regard to valvular diseases; it is the key to the most important part of the pathology of the heart. (*This last conclusion is certainly the least substantial of all the deductions made by Hope.*)

It must be candidly acknowledged, that this part of diagnosis has been very much neglected in France. It is rarely that elementary works contain any remarks on the means of recognising the orifice or the valve affected. Hope has, especially in the third edition of his valuable work on the *Diseases of the Heart*, fixed the basis of this science, and consequently, has given to diagnosis more certainty than was anticipated for some years. As will be properly supposed, we make a liberal use of the researches of this judicious observer.

The murmurs which take place in the heart, at whatever orifice produced, will be heard better over the ventricle corresponding to the alteration, than over the opposite ventricle.

The murmurs caused by lesions of the left ventricle are best heard over the left side of the chest, outside of the heart; those which have their seat in the right ventricle are not heard beyond the limits of the præcordial region, but are perceived at their maximum over the whole line of the synchondro-sternal articulations, the inferior part of the sternum, sometimes even in the epigastric region; it is an error to say, that these murmurs are prolonged over the right side of the chest.

Murmurs which are seated at the arterial orifices, are at their maximum immediately above the semilunar valves, that is, on a level with the inferior border of the third rib near the sternum, when the patient is in a horizontal position; and a little lower when he is standing up; thence following the course of the aorta or the pulmonary artery for the space of two inches.

It will be easily recognised that a murmur belongs to the aorta and not to the pulmonary artery, because the murmur is distinctly heard by tracing the course of the first vessel, whilst it is very full, and scarcely audible, over the course of the last.

In conclusion: a murmur will be discovered to depend rather on an alteration of the valves of the aorta, than on a disease of the ascending aorta, by its diatonic key, which does not commonly exceed the sound produced by the repetition of the letter R: whilst that produced by this last cause is much higher, *nearer*, more super-

ficial, and presents much analogy to the sound produced by the repetition of the letter S.

Everything which has been just said is equally applicable to the pulmonary artery. It may be well, however, to add, that the murmurs referrible to the valves, or the artery itself, are always more superficial and nearer than those of the aorta, in consequence of the more superficial situation of this first artery, and the more elevated position of its orifice.

In order to ascertain the seat of the murmur, it is only necessary to ascertain the course of the aorta or pulmonary artery, which will be found entirely beyond the circle in which the murmurs of the auriculo-ventricular orifices are formed. This rule applies not only to the contraction of the arterial orifices, but also to their defects, with this restriction, that these murmurs of regurgitation are more feeble, and are not so easily transmitted along the course of these vessels.

The murmurs which are placed at the auriculo-ventricular orifices, are best heard over that portion of the præcordial region where the heart is in contact with the sides of the thorax, where there is dulness on percussion, in other words, about the apex of the heart. It is towards the upper and left side of the dull region, about the fifth rib, or the subjacent intercostal space, a little to the right of the nipple, that the maximum point of the murmurs belonging to the auriculo-ventricular orifice may be found: whilst those of the right auriculo-ventricular orifice are better perceived at the upper and right portion of the dull part, that is, over the sternum, or a little to the side of this bone. At these two points, the murmurs of the two orifices are very near and very distinct, whilst those of the arterial orifices are always obscure and distant when you look for them about the apex of the heart. The murmur from regurgitations through the semilunar valves, which descend a little towards the inferior part of the ventricle, must be excepted. But these regurgitant murmurs always increase in intensity in proportion as you ascend towards the arterial orifices, whilst, under the same circumstances, the murmurs of the auriculo-ventricular orifices always diminish in intensity.

When the murmurs take place simultaneously at the arterial and auriculo-ventricular orifices, it is sufficient, in order to insure the diagnosis, to remember the preceding rules, and the tones of the murmur, so as to be confident that there are two distinct sources of these sounds.

In conclusion: when two murmurs are seated in the same orifice, it is easy to be satisfied of the fact; first, by their maximum point, and secondly, by their intimate connexion with the first and second sound.

2d. *Vibratory Tremor.* — In certain persons, the application of the hand or the ear over the præcordial region, renders perceptible a species of rustling sound. This rustling sound, noticed for the first time

by Corvisart, was by Laennec compared to the purring sound which cats make when fondled; but M. Bouillaud much more properly compares it to the trembling which is experienced when the hand is placed on the larynx of a man who is singing. It transmits to the ear the sensation of repeated vibrations. There is in this vibratory tremor of the heart two things to be considered; the *tactile part*, or the vibrations, which are created in the interior of this organ, and have sufficient strength to propagate themselves to the sides of the chest; and the *acoustic part*, that is, a roaring noise more or less prolonged, with distinct and more or less near sounds. It is only requisite to listen for a few moments to this tremor, to be assured that it is caused by solid and resisting obstacles, which are set in vibration by the passage of the column of blood. This was the opinion of Corvisart, who referred the cause of it to ossification of the mitral valve. Facts have confirmed this theory; it may now be regarded as settled, that every vibratory tremor which is observed towards the heart, is connected with some organic lesion, consisting in some obstacle which resists the circulation of the blood. This species of tremor is really nothing more than the highest degree of the phenomena which we have just studied under the name of valvular murmurs, and of other phenomena, which we shall soon investigate under the head of "*friction sounds;*" all three are the result of a greater or less amount of friction, and are often confounded with each other. It is necessary, in order to produce a vibratory tremor, that the vibrations which result from the passage of blood over the obstacle, be at once numerous and very continued, and besides, that agents of transmission be found in the adjacent parts.

It will not suffice for this tremor to take place in the interior of the chest only; it must be propagated outwardly; in certain cases, the hand applied over the præcordial region could scarcely feel a tremor, whilst the ear recognised numerous vibrations; therefore this propagation is subordinate to the seat of the tremors.

It is rare that this tremor is found depending on a disease of the valves or of the aortic orifice, because the sternum, which is placed above this orifice, opposes this transmission; but if, by chance, the aorta becomes displaced, there is then perceived a vibratory tremor, when the aortic orifice is diseased. The same cause operates to prevent our admitting this tremor to depend on a disease of the orifice of the pulmonary artery, or of this artery itself. In certain cases, if the invalid incline to the left side, this phenomenon becomes perceptible between the second and third left ribs. In the case of dilatation of the pulmonary artery, there is, on the contrary, a very distinct tremor at this point. But of all the causes giving rise to this tremor, the most frequent is that which Corvisart had observed, viz., the deficiency of the auriculo-ventricular valves, and especially of the mitral valve.

It might be mentioned, *à priori*, what are the material conditions of this tremor. Theory indicates, and observation proves, that it

is owing to a solid obstacle projecting in the course of the column of blood. It is thus, that in the aorta and pulmonary arteries it is due to the presence of *bourrelets*, projecting osteo-cartilaginous tubercles; it is also in this manner that, at the left auriculo-ventricular orifice, the vibratory tremor is most often caused by a peculiar alteration of the mitral valve, consisting in a considerable elongation of the free extremity of this valve, which forms, in the interior of this ventricle, an elongated projection, and pinged like a purse.

As this phenomenon is the result of the percussion of the blood against a solid obstacle, it follows, as a necessary consequence, that it is entirely subservient in its production to the intensity with which the column of blood strikes against this obstacle, and to the greater or less energetic contractions of the heart.

We have already remarked, that one of the lesions which most often gives rise to this tremor is the deficiency of the mitral valve; since, under these circumstances, the blood penetrates into the auricle with all the force which is communicated to it by the general contraction; whence it follows, that the vibratory tremor ought to be very rare in the case of simple contraction of the auriculo-ventricular orifices. In relation to the arterial orifices, it is clear, from the same principles, that the tremor which is the result of a contraction of these arterial orifices, will possess much more intensity than the tremor resulting from a deficiency of the corresponding valves. Whatever be the cause of the tremor, it diminishes and disappears completely when the contraction of the heart is enfeebled, when the vital forces commence to fail. It would be a grievous error, to consider the disappearance of this phenomenon as an evidence of amelioration of the patient's state; it informs us, on the contrary, that it is considerably aggravated, and that the circulation does not take place with the force and regularity necessary to the proper accomplishment of the functions.

The vibratory tremor may be advantageously employed in the diagnosis of the diseases of the valves; it answers well to apply to it the rules that have been given on the subject of valvular murmurs; the point where this tremor is best heard must be sought for; its elevation in the musical scale must be studied; it must be determined with what movement of the heart it coincides: the *systolic* vibratory tremor announces either a deficiency of the mitral or tricuspid valve, or a contraction of the arterial orifices; in the first case, this tremor has its maximum of intensity on a level with the point of the heart, or a little higher; in the second case, it is at its maximum on a level with the arterial orifices; that is, in the third intercostal space. When this tremor is double, it may indicate a contraction of an arterial orifice, with a deficiency of the mitral or tricuspid valve, and *vice versâ;* or a contraction with deficiency of an arterial or auriculo-ventricular orifice. In this last case, it is necessary to find at what points this double tremor has its maximum of intensity, and whether it has not two sources of production (which is excessively rare, if it ever has been observed).

In finishing whatever relates to the vibratory tremor, we say, that the friction sound of which we intend to speak, soon puts on, under certain circumstances, the character of the vibratory tremor, and gives rise to quite analogous vibrations. It is at the beginning or end of pericarditis, when the false membranes are dry, or the folds of the pericardium, rendered rough by the presence of false membranes, churn, as it were, between them a certain quantity of liquid, that vibrations similar to those of the tremors are observable. It is proper to be aware of this circumstance, although the distinction is sufficiently easy, because the symptoms that are observed in these last cases are those of pericarditis, and not those of chronic endocarditis, nor of valvular diseases.

Peripheric or Pericardiac Friction Sounds. — In the healthy state, the friction of the two folds of the pericardium is not accompanied by any perceptible noise; this is not the case, however, when the friction is increased in intensity, either by the acceleration of the motions of the heart, or by the more or less rugose surface of the folds of this membrane; for then, each rubbing together of the opposite folds of the pericardium is accompanied by a particular sound, to which the name of *peripheric* or *pericardiac* friction has been given. This phenomenon ordinarily coincides with the systole and diastole of the heart. It is almost immediately recognised by the ear of the observer, and rarely exceeds the limits of the præcordial region; it never ascends up the course of the great vessels. This friction sound is most distinctly heard on a level with the apex and inferior portion of the heart for the systole, and at the middle portion and base for the diastole.

It is not thus, when the conditions of the opposed surfaces of the pericardium are not similar in their whole extent; it is, in this last case, on a level with the roughest portion that this peripheric sound is at its maximum.

During palpitations, the force and rapidity with which the pulsations of the heart take place, often cause the production of a double friction sound, which is superficial, having a peculiar dry character, and may best be compared to the sound produced by passing the hand rapidly over cloth; this phenomenon always preserves this character. It is otherwise in pericarditis, of which the friction sound constitutes one of the most essential phenomena; in this disease, this sound is capable of putting on different characters; dull, grave, deep, at first, it becomes more dry and crackling in proportion as the disease advances. The numerous modifications which this friction sound experiences, have enabled authors to create a great number of anomalous sounds, which they have described under the name of *friction sounds*, or like the *grating of nutmeg*, *creaking of new leather*, or *rumbling*.

All these sounds are merely modifications of one and the *same sound*, viz., the *friction sound*. If these anomalous sounds do not deserve to be distinguished into so many independent phenomena, they all have their value, since they correspond to certain states of

the serous surfaces of the pericardium. The determination of these morbid states has not been made in a satisfactory manner; however, it may be said, in general, that the dry and crackling character of the friction sounds is much more marked in proportion as the false membranes require a greater density. Hope, who has investigated this subject with much care, has arrived at these conclusions:

1st. That the *rasping* and *filing* sounds are produced by the friction of firm and rough false membranes.

2d. That the *rustling* and *crackling* sounds are owing to pseudo-membranous surfaces which are soft and humid, whilst the

3d. The *softer rustling* sound, is ascribed to soft and rather dry lymph.

4th. That the *creaking* and *croaking* sounds are present where very dry, tough lymph is met with.

5th. That the *continuous rumbling* sound is due to the agitation of a small quantity of liquid between the folds of the pericardium, covered by lymph.

Without attaching too much importance, either to the comparisons which have been made of these different sounds, nor to their ascribed mode of production, we are of opinion that the preceding remarks will be useful in the diagnosis of pericarditis.

The seat of the false membranes exercises a certain influence over the production of the friction sound; it is thus, that where the plates of false membrane only occupy the base of the heart, and, consequently, a part where the friction is always very feeble, the anomalous sound does not exist.

The friction sound, characteristic of pericarditis, which, as we have seen, follows in the train of the disease, appears to change its place from day to day, under the ear of the observer; this announces the fact, that the inflammation is extending and increasing.

Like all the anomalous sounds, this phenomenon bears a relation to the frequency and energy of the pulsations of the heart; all those causes (such as palpitations) which increase this frequency and this energy, impart greater strength and a more roughened character to the friction sound; whilst the causes which depress the action of the heart (such as bleeding), or which render it slower (as digitalis), impress on it quite opposite characters.

4th. *Sound of the impulse of the apex of the heart against the thoracic parietes*, or *the metallic tinkling sound.* This sound, which Laennec pointed out in nervous palpitations, consists in a metallic clacking of more or less intensity, of which an idea may be formed by applying the palm of the hand on the ear, and tapping lightly the back of this hand with the finger of the opposite hand (*auriculo-metallic sound* of M. Filhos).

This sound, which is only perceived during the systole and never during the diastole, and which immediately follows the first sound, depends on the impulse of the heart against the inferior border of the fifth rib. The proof of this is, that it can be made to cease by

the will, by pressing the soft parts into the corresponding intercostal space, so as to put the internal parts of this space on the same level with the rib, upon which the heart glides without striking. This metallic tinkling is only noticed in lean subjects, because in subjects of a robust constitution and well formed, the intercostal spaces are full and resisting; and consequently the border of the fifth rib is not exposed to the impulse of the apex of the heart. However, in some cases, this metallic tinkling appears to be owing to the motions which the violent contractions of the heart communicate to the liquids and the gas which distend the stomach. The production of this phenomenon depends always on the force and frequency of the pulsations of the heart; all the causes which render these pulsations stronger and more frequent may induce the metallic tinkling; this anomalous sound does not deserve all the importance that M. Filhos has given to it, for it only modifies the sounds of the heart so as to render them not easily known again, and is as yet useless to diagnosis.

B. *Anomalous Sounds from Inorganic Causes.*

The anomalous sounds belonging to this head are those which are not caused by any alteration of the solids, which constitute the different parts of the heart and its appendages.

Whilst the anomalous sounds from organic causes take place in the interior of the heart, or exterior to it, the anomalous sounds from inorganic causes all have their seat in the interior of this organ, and thence are reflected on the large vessels which arise from it.

The illustrious author of auscultation had recognised the fact, that anomalous sounds are observed in the neighbourhood of the heart, which could not be accounted for by any organic lesion after death; rejecting altogether the idea that these anomalous sounds could be owing to any state of the blood, or to the manner of its movements, he quickly renounced the conclusion which he had previously arrived at, respecting the nature of the bellows sound in the first edition of his work; he concludes, that it was evident that the bellows sound, like the vibratory tremor, is caused by a true spasm of the heart, a disturbance of innervation.

Let us first examine under what circumstances this bellows sound from an inorganic cause is found; it is always in subjects who have been submitted to considerable losses of blood, or who have had hemorrhages; and also in persons subject to cachectic states of the system, such as chlorotic, cancerous, tuberculous, scorbutic cachexies, &c., that is, in cases where the sanguineous fluid, if it has not diminished in quantity, has at least lost much of its natural richness; and finally, in certain temporary pathological conditions, which are accompanied by great disorder in the circulation, in the chills of intermittent fevers, for example, as M. Gendrin has shown. Thus, then, diminution in the quantity of the liquid, diminution in

the plasticity of this liquid, are in general the circumstances under which the anomalous sounds of the heart from an inorganic cause are produced.

The anomalous sounds, when they are seated in the heart (they are generally more marked in the large vessels), deserve the name of bellows sound; they always present a soft and rumbling sound; they give to the ear the sound which is produced in blowing a candle. Always synchronous with the systole and *never* with the diastole of the heart, they always have their maximum of intensity on a level with the arterial orifices, and often extend along the course of the large vessels; so that it is only necessary for the bellows sound to be synchronous with the diastole or the second sound, and to have its maximum on a level with the apex of the heart, in order to be convinced that it depends on an organic cause.

Like all the other anomalous sounds, the bellows sound from an inorganic cause is in proportion to the force and rapidity of the circulation. In palpitations of the heart, the inorganic murmur increases so as to become vibratory; whilst in the last moments of life, when the heart has not sufficient energy to contract, these sounds cease to be perceptible.

It now remains to find out the immediate cause of this inorganic bellows sound; the experiments of Spittal, Hope, Piorry, Bouillaud, &c., in accordance with the laws of acoustics, establish the fact, that when a liquid is made to circulate in a tube without admixture of air, a sound is produced which varies in intensity, according to the rapidity with which the liquid is propelled in the tube, whence it follows that the sound is in a direct ratio with the intensity of the friction. The question then presents itself: in the case of bellows sound from inorganic causes, is there an increase of friction in the heart and arteries? We reply in the affirmative. What are really the conditions in which this species of bellows sound is produced; is it not when the quantity of blood is diminished, or when this liquid has lost its plasticity? In the first case, is not the column of blood, having lost in weight, thrown with more rapidity against the walls of the heart? Moreover, does not the state of flaccidity of the arteries favour the production of vibration in the parietes of these vessels? Does not the diminution of the viscosity of the sanguineous molecules, resulting from the impoverishment of the blood, favour the gliding of these molecules on each other, and consequently their collision against each other, so as to produce vibrations? It should be noted, also, that the state of irritability of those persons whose systems manifest a considerable alteration in the quality or quantity of blood, imparts to the contractions of the heart the character of roughness and rapidity, whence there is greater friction, and consequently vibration of the walls of the vessels. We think, then, that the inorganic bellows sound of the heart is due to three causes: — 1st, the impoverishment of the blood; 2d, the state of flaccidity of the arteries which arise from this organ; 3d, the rapidity with which

the column of blood circulates. There may be added, as an auxiliary cause, the oblique position which the large arteries contiguous to the heart present to the column of blood that springs from this organ at the moment of its contraction; a circumstance which gives rise to currents and counter currents on a level with these orifices, and consequently to sonorous vibrations.

To resume: The sounds of the heart may be modified by anomalous sounds, both organic and inorganic: of the *first*, some, as the *infra-cardiac murmurs* and the *vibratory tremor*, take place in the interior of the heart; others, as the *superficial friction sound*, and *metallic tinkling*, are seated exterior to the heart. The *second*, on the contrary, are all found in the interior of the heart, and possess the characters of soft murmurs, which never become vibrating. How are we to distinguish one from the other? By the following signs:

1st. The *inorganic murmurs* are always simple, whilst the *murmurs from an organic cause* may be double.

2d. The *inorganic murmurs* always have their maximum on a level with the arterial orifices; the *organic murmurs* may occupy all the orifices.

3d. The *murmurs from an inorganic cause* always have the character of a soft bellows sound; they never acquire the rough character which may be exhibited by the *organic murmurs*.

4th. The *inorganic murmurs* often extend with increased strength along the course of the large vessels; the *organic murmurs* only extend under certain circumstances, and always with less force.

5th. The *inorganic murmurs* are not permanent; they disappear under the influence of rest and good regimen; the *organic murmurs* persist indefinitely.

CHAPTER IV.

NORMAL AND PATHOLOGICAL STATES OF THE PULSATIONS AND SOUNDS OF THE ARTERIES.

SECTION I.

OF THE ARTERIAL PULSATIONS IN THEIR PHYSIOLOGICAL AND PATHOLOGICAL STATES.

At the moment when the column of the blood, impelled by the systole of the heart, enters the arteries, it produces an elevation or mechanical impulse, which bears the name of *pulse*, or *arte-*

rial diastole; after which the artery returns manifestly to its former condition (*arterial systole*). Each arterial diastole corresponds exactly to the systole of the heart, allowance being made for the short space of time which the column of blood, propelled by the heart, takes to arrive at the artery under examination. Each arterial pulsation is accompanied by an increase in the length and volume of the artery. The elongation of the arteries is proved by the increase of the curvature of these vessels, at the moment of the passage of the column of blood: as may be easily seen in the superficial arteries, as the brachial and temporal. The most simple reasoning is sufficient to admit the increase of the volume of the arteries, at the time when a new quantity of blood impels, *à tergo*, the blood already enclosed in the arterial vessels; and the elastic structure of these vessels tends to confirm this opinion. It, therefore, follows that the arterial beats which form the pulse, consist, 1st, in an increase of the curvature of those vessels, a result of their elongation; 2d, in an augmentation of their volume.

The arterial pulsations are immediately under the influence of the heart's action. In the healthy state, the beatings of the pulse are a faithful index of the rhythm, frequency, force, duration, &c., of the systoles of the heart; and when the arteries are healthy, it may be said, in a general manner, that the intermissions, the irregularities, the increase or diminution in the frequency and force of the arterial beats, represent so many analogous conditions in the systoles of the heart. This is not the case in certain circumstances, and especially when the arterial trunks are diseased. It is true, that then the localization of the phenomenon, and its circumscription to only one portion of the tube, are sufficient to guard us from error.

As a general rule, while the contractions of the heart preserve their force, the pulse is a faithful representative of the state of these contractions, and principally those of the left ventricle; but when the organ is excessively enfeebled, the pulse cannot be felt in the arteries distant from the central circulatory organ. We may be easily convinced of the cases in which there is extreme slowness of the circulation, if, by applying the hand over the præcordial region, two or three systolic impulses of the heart are felt corresponding to one impulse of the radial artery. There is then a discordance between the pulsations of the heart and those of the arteries.

The pathological alterations which interfere with repletion of the sanguineous tubes, produce another discordance between the pulsations of the heart and arteries: so that, when the blood, whether from considerable diminution of the arterial orifices, or from obstacles to the auriculo-ventricular orifices, does not penetrate in sufficient quantity into the ventricular and arterial cavities, the heart, however energetic may be its contractions, transmits into the arteries only a very small quantity of blood, which

scarcely raises the finger. The pulse is *feeble*, and contrasts by its feebleness with the energy of the systoles of the heart; at the same time it is irregular, in consequence of the irregularity of the contractions of the heart itself. It is especially in the contraction of the left auriculo-ventricular orifice, and principally in deficiency of the mitral valve, sometimes, also, in a considerable diminution of the aortic orifice, that this feebleness of the pulse with irregularity is observed. In the disease which bears the name of regurgitation by the aortic valves, and in which the repletion of the arteries is so much impeded, the pulse, without losing its regularity, becomes quick, short, and *jerking;* but, at a more advanced period of the disease, the arteries become hypertrophied in order to accelerate the circulation, and the arterial diastoles are lengthened, so that the artery is not depressed after each diastole, which appears as if it were only a simple increase of the preceding diastole. (Gendrin.)

The alterations in the quality and quantity of the blood exert no less influence over the state of the pulse. Thus, in anemic subjects, the arterial beats are very feeble, and thereby contrast with the suddenness of the impulse of the apex of the heart: in subjects liable to cachexiæ (chlorosis, for example) the arterial diastole is sudden, energetic, as it were rebounding, but compressible; in plethoric persons, the arterial pulsations, ordinarily full and large, may present quite opposite characters; and even the diastoles may appear very feeble in consequence of the violent distension which the vessel has already undergone. This is a false feebleness, by which one must not be deceived; the injection of the skin, the energy of the contractions of the heart, are the signs by which this condition may be recognised. If you bleed, under these circumstances, the pulse will be seen to rise, and the congestions will disappear; whilst, if expectant remedies be employed, the plethora will continue to increase, and the congestions will be converted into true inflammations.

The diseases of the arterial tubes, which often coincide with those of the heart, create, in addition to the modifications produced by the state of this organ and the blood, special alterations of the pulse. Slight diseases of the arteries cause them to lose, in a great measure, their elasticity; consequently, the pulse is quick and short, without increase in the volume or the diameter of the vessel; the pulsations are scarcely perceptible, but there is often felt a kind of tremor.

To recapitulate: the arterial pulsations may be modified in their force and rhythm, not only by causes which depend on the heart, but also by causes which depend on the quality of the blood, as well as on the arterial canals which this liquid traverses. As it is very rare to find a general disease of the arterial tubes, this will enable us to distinguish the modifications of the arterial pulsations which depend on alterations of these vascular tubes.

SECTION SECOND.

OF THE SOUNDS OF THE ARTERIES IN THE HEALTHY AND MORBID STATE.

When, in a healthy person, an artery of a certain size is examined by the ear or the stethoscope, at each arterial diastole there is felt a sensation of rising, with a slight, and not very clear, indeed almost dull sound. It is not uncommon to hear, in some cases, after this first dull sound, which belongs to the artery, a clearer one, which appears to be distant: this is the second sound of the heart, transmitted from point to point, through the medium of the walls of the arteries, by the column of blood which traverses them. It sometimes happens, that in the arteries near the trunk, as the carotids, and especially in the left, there are observed anomalous sounds, which are, in fact, only the prolongation in the arteries of those which take place at the orifice of the aorta; and hence the reason why these murmurs are observed to increase in proportion as you approach the heart, and why they are always stronger in the left carotid than in the right. The reverse obtains in the diseases of the arteries, as well as the murmurs from inorganic cause.

The arterial sound, or diastolic sound of the arteries, is synchronous with the pulsation of these vessels, and is repeated at each passage of the column of blood. It varies in intensity, and other respects, in obedience to a number of conditions; but, in general, it bears a relation to the diameter of the vessel in which it is produced. Dull in the arteries which are full of blood, it becomes less so, and resembles the blowing sound, when the artery is slightly soft and flaccid: it is more intense in lean than in fat persons, and its increase in intensity is facilitated by the tension of the parts concerned. The more forcible the compression made by the ear or stethoscope, the more clear is the sound. In some persons (but not in all, as almost every author has remarked), the bellows sound may be artificially produced.

The opinions which we have expressed, concerning the cause of the anomalous sounds of the heart, from inorganic causes, save us the necessity of insisting, at length, on the mode of production of the healthy sound of the arteries. It is evident that it belongs to the same class; and is simply the result of the friction of the blood against the arterial parietes, to which are added the vibrations produced by the curves of the bloodvessels, and by the prominences which are situated at each division of these vessels; the whole being increased by the pressure of the ear or stethoscope of the observer. The normal sounds of the arteries, like those of the heart, may be modified in extent, force, tone, and rhythm; but so little attention has been paid to all these modifications up

to this time, that it is impossible to establish as yet anything satisfactory in relation to the diagnosis. The only modifications which are known, consist in the replacement of these normal sounds by murmurs of a different kind. We shall divide the anomalous sounds of the arteries into *organic* and *inorganic*, or those proceeding from organic and inorganic causes, as was done in the study of the sounds of the heart.

A. *Organic anomalous Sounds of the Arteries.*

1st. *Murmurs of the arteries (intermittent blowing sound, friction sound of the arteries).* Whenever the arterial tube acquires a great consistence; when osseous or cartilaginous plates occupy the coats or contract its diameter; or an aneurismal tumour, on the side of its canal, communicates with the interior of the vessel; when the vessel itself is considerably dilated; in fine, whenever the diameter of an artery is much contracted or increased, the dull and flat sound which has just been mentioned is no longer heard; but at each arterial diastole a true murmur is heard, whose nature and intensity are subject to various modifying circumstances. The murmur from organic cause is always intermittent; it usually coincides with the systole; but in some rare cases of arterial aneurism, a double blowing sound is heard, both systolic and diastolic, separated from each other by an appreciable interval, and both having their maximum of intensity on a level with the opening of the aneurismal sac. This sound, like all anomalous sounds, is in a direct proportion to the depth of the artery, the rapidity of the circulation of the blood, the more or less dense state of the parts which separate the arterial tube from the ear of the observer, and finally, the pressure which he makes with the stethoscope. The nature of the cause impresses an equal amount of variation on this anomalous sound. If there be contraction, this sound is more acute; if the vessel is dilated above this contraction, as often happens, the intensity of the murmur increases; if the obstacle be cartilaginous or osseous, the sound is dry; whilst it is obscure and dull, if this obstacle be soft and fleshy. Sibilant under certain circumstances, (for example, in considerable contraction of the vessel, or in aneurism, with a more or less narrow aperture,) it becomes rough, blowing, and vibratory when the rugosities which are scattered over the surface of the vessel are moveable and pediculated. The murmur from organic causes is better heard in arteries of a large size.

2. The *purring tremor of arteries from organic causes*, which we are naturally led to investigate, presents itself in the same conditions of system which cause the organic murmurs of the vessel. It is only necessary that the pathological conditions of the artery be such as to cause sonorous vibrations, appreciable by the hand

and ear. Thus, in aneurism of the aorta, in cases of considerable induration of the arterial tubes with rugosities on their internal surface, of sacciform dilatation of some arteries, as the aorta or pulmonary artery, a very marked vibratory tremor is perceived. This tremor of the arteries always coincides with the arterial diastole; the circumstances which favour its extent are the same as for the arterial murmurs. This phenomenon is generally very limited; the more or less rough murmur which accompanies it extends to a greater or less distance.

B. *Anomalous Sounds of the Arteries from Inorganic Causes.*

1st. *Arterial murmurs.* — When, in certain persons, the ear or stethoscope is placed over the large arteries, as the carotid or the subclavian, an anomalous sound is perceived, which presents different varieties. Sometimes it is a tolerably clear bellows sound, like that produced by the instrument which has this name, its intensity bearing a relation to the vessel in which it is seated, and to the rapidity of the contractions of the heart, and coinciding perfectly with the arterial diastole (*intermittent bellows sound*). Sometimes, the bellows sound is double, and of the two sounds constituting it and which succeed each other without hardly any appreciable interval, the first, which coincides with the arterial diastole, is much stronger than the second (*bellows sound with double currents*); sometimes, this murmur is continuous, and analogous to that of the sea, or to the sound which is heard when the ear is applied near a large univalve shell fish; and this continuous murmur may be circumscribed by the caliber of the artery, or may present a diffused character (*continuous murmur* of Laennec). Occasionally, this continuous murmur presents a higher degree of elevation; it is transformed into a sort of humming sound, more or less musical, analogous to that which the humming top, or the plaything of children, to which they have given the name of devil, produces (*bruit de diable* of M. Bouillaud). This humming rises up always gradually to its middle point, in a manner synchronous with the arterial diastole. Sometimes, finally, this murmur, rising still a degree higher, produces true arterial tunes, in the sense in which we understand them, in the midst of the continuous murmurs, consisting of many notes, separated by easily recognisable diatonic intervals. Those musical sounds may, with some degree of exactness, be compared to the sound produced by a metallic cord just struck, or by that which the motion of the great fly, and that of the boiler at the commencement of ebullition.

All these sounds are, in some measure, only different degrees of one and the same phenomenon, viz., the intermittent murmur which pa ses into the continuous murmur. There is only one step from the more or less obscure continuous murmur to the humming and

musical one; some slight modifications in the rapidity of the current of blood, so as to press upon the vessel, being all that is requisite in order to induce these successive transformations.

It is ordinarily on the lateral parts of the neck in the carotids and subclavian, and especially on the right side, that these arterial murmurs are at their maximum. It does not appear that the larynx or the act of respiration, has all the influence which has been ascribed to them by some observers over the production of this phenomenon; and, besides, murmurs of this kind may be detected in other arteries than the carotid and subclavian (as in the abdominal, the crural, brachial, &c.).

2d. The *purring or vibratory tremor of the arteries*, which is an occurrence of the same order as the preceding, coincides often with the inorganic arterial murmurs, and especially with the musical bellows sound; the vibratory tremor perceived under these circumstances, is a little more diffused than the arterial one from organic causes; sometimes it is limited to a certain number of arteries (those of the neck, for example); at other times it occupies all the arteries of the body.

It is now generally admitted as a fact, that the murmurs and purring tremor of the arteries from inorganic causes are only met with in persons whose blood presents alterations, either in its quantity or quality (deficiency of the valves of the aorta, anemia, cancerous and scorbutic cachexiæ, &c.), and also in certain pathological states, which are accompanied by a concentration of the blood (paroxysms of intermittent fevers). These being the conditions in which they are found, let us inquire how they are produced.

In speaking of the murmurs of the heart from inorganic causes, we have already shown, that the alterations in the quality or quantity of the blood increase the friction against its parietes. In the arteries the circumstances are still more favourable. It is thus, that in anemia and deficiency of the aortic valves, the vessels when empty of blood return to their former state; and independently of their flaccidity, there are folds formed in the internal surface of these vessels; and hence, an increase of friction. Let it be added, that, under the influence of losses of blood, the circulation is constantly accelerated to the amount of fifteen to twenty pulsations per minute; and that there is in the arteries, on a level with all the branches that spring from a main trunk, a prominence, which, projecting into the arterial cavity, breaks the current of blood, and causes sonorous vibrations. In certain cachexies, where the blood is more deficient in quality than in quantity, the molecules of this liquid, being lighter and less viscid, and consequently easier of motion, pass more rapidly over the internal membrane of the arteries. To this cause may be added the flaccid state of the arteries, the rapidity of the contractions of the heart, &c.

In this manner the intermittent murmurs of the arteries, and their purring or vibratory tremor, which is only an augmentation

of the former sound, are explained; but it is not the same as regards the murmurs produced by a double current.

We have placed the seat of the continuous murmur in the arteries, but the labours of Dr. Ward, of Birmingham, and of Hope, have caused doubt on this point.

If it be considered that the continuous murmurs persist during the systole, and almost with the same intensity, that is, at a moment when the column of blood obeys no more than this feeble elasticity of the arterial tissue; that when the pressure is increased with the stethoscope, they give to the ear a sensation of a bellowing or rumbling sound; that they are commonly diffused, and not circumscribed, by the caliber of the artery; that these continuous murmurs are constantly reinforced at each arterial diastole; that they seem to the ear like two currents flowing in different directions; that it suffices, under some circumstances, to press lightly above the stethoscope, in order to cause them to cease, and only to allow the sound of the arterial diastole to be heard, we should join Dr. Hope in the inference, not that all the bellows sounds are seated in the veins, as MM. Barth and Roger have imagined this author to say; but rather, that in the continuous murmur there are two things, viz., the venous element, if we may be allowed the use of the expression, which constitutes the foundation of the anomalous sounds, and which itself is continuous like the venous circulation; and the arterial element, which reinforces the continuous sound at each arterial diastole. In a work which we shall soon publish, we will mention some curious facts and experiments, which appear to us to sanction this opinion: at present, we only intend to present the difficulty.

In summing up, we find that the arterial sounds may be modified by organic and inorganic anomalous sounds. We now present their distinctive characteristics.

1st. The *arterial murmurs from an inorganic cause* may be double; *those from organic causes* are almost always simple, and seldom take on the continuous character.

2d. The *inorganic arterial murmurs* are best observed in certain arteries (as the carotid and subclavian); the *organic arterial murmurs* have no fixed position.

3d. The *arterial murmurs from inorganic causes* always have a soft character; they are never rough like the *organic murmurs.*[1]

4th. The *inorganic arterial murmurs* are not permanent; they increase during the heart's palpitations, and disappear by rest, and under the influence of animal food; the *organic arterial murmurs* are permanent, like the cause which produces them.

The *vibratory tremor from an inorganic cause* may be distinguished from that proceeding *from an organic one*, 1st, by the character of the murmur which accompanies it; 2d, by its being of less intensity; 3d, by its diffused character, and its propagation over a large extent of the arterial system; 4th, by its want of permanence.

SECOND PART.

PATHOLOGY OF THE HEART.

CHAPTER I.

INTRODUCTORY OBSERVATIONS.

General Considerations on the Symptoms of the Diseases of the Heart — Rules to follow in the Examination of these Diseases. — Classification.

If we were to conform to general custom, we should notice in this place the causes, symptoms, complications, and treatment of diseases of the heart generally; but, reflecting on the little knowledge which is possessed in regard to many of these points, we are convinced that it would be impossible, in the present state of the science, to give true general views, without exposing ourselves to the risk of falling into one of the dangers pointed out by Bichat, either of generalizing or particularizing too much.

Let it suffice, therefore, at present, to cast a rapid glance over the prominent symptoms of diseases of the heart.

This short review will permit us to give some details on a number of symptoms which supervene sooner or later in the diseases of the heart; and principally on those which depend on obstructed circulation. We shall thereby avoid repetitions, which we could not otherwise escape, in tracing the special pathology of the heart. It will be sufficient to cite only one symptom, in order to make the reader acquainted with its nature, mode of production, &c. We shall then try to exhibit the chain of symptoms; in other words, to determine the modifications which are operative in cardiac diseases at their different periods.

Like all diseases of important organs, those of the heart present both local and general symptoms; the local are composed of all the signs furnished by inspection, the touch, and mensuration, as *palpitations*, *prominence*, &c.; or by auscultation and percussion, as the anomalous sounds of the heart and arteries, and dulness. We have given sufficient space to most of these signs in preceding chapters, and therefore will not speak of them here. The dulness of the præcordial region, and the modifications which it presents, merit our attention for a few moments. It is a well known fact, that if you strike solid bodies like the heart, a dull sound is obtained, whilst a clear sound is heard, when you strike bodies which contain air, as the lungs and stomach. In the healthy state,

the percussion of the præcordial region gives, within a line, which, starting from the synchondro-sternal articulation, ends at the left hypochondriac region, five or six centimetres from the middle of the xiphoid appendix, a dull sound, limited externally by the preceding line, internally by the left border of the sternum, superiorly by the synchondro-sternal articulation of the second rib, and inferiorly by the apex and the inferior border of the heart. In an adult of medium height, and whose heart is well proportioned, this dulness, or obscure sound, has a diameter of an inch and a half to two inches. Its modifications in the præcordial region resulting from diseases of the heart, are of the highest importance in diagnosis. This dulness may be augmented or diminished. 1st. The increase in extent of the dulness of the præcordial region may depend on an increase in the volume of the heart, as in hypertrophy or dilatation, fatty developments, congestions of this organ, or, also, from a considerable accumulation of liquid, false membranes, or lymph in the serous cavity of the pericardium. In the former case, it extends principally downwards; the apex of the heart is displaced very much, downwards and outwards: in the latter case the dulness is much more complete; it occupies all the region of the heart, even to the second rib; and when the position of the apex of the organ is found, there is no observable displacement. The dulness of the præcordial region may be increased by causes independent of the heart itself; thus, it may be augmented superiorly by tumours of the mediastinum, by aneurisms of the aorta or pulmonary artery; or, when the anterior border of the left lung is indurated, by inflammation, tubercles, &c. 2d. The diminution of the dulness of the præcordial region may depend on a diminution in the volume of the heart, or its atrophy; but more commonly it results from emphysema of the anterior border of the left lung, which is recognised by tympanitic sound of the præcordial region, by the feebleness of the beats and sounds of the heart, and especially by the recognition of the vesicular murmur at this point.

General Symptoms. — The diseases of the heart exert a very marked influence over the whole economy; nor is it in a narrow and circumscribed circle that these morbid reactions are produced; but, on the contrary, how numerous are the sympathies which the central organ of the circulation creates in the rest of the organism! It is in consequence of their multiplicity, and the difficulty of referring them to one perfectly settled cause, that we have decided to study the general symptoms in a purely analytical order:

1st. *Congestions.* — The embarrassment of the circulation is marked in the skin, and principally in the face; sometimes by a violet and livid discoloration, general or local; sometimes by a livid paleness, which has a frightful appearance. It is not an uncommon event, to see the face of a blue colour in its whole extent; but it is much more rare to see all the body present this colour, except in certain congenital diseases of the heart. The

engorgement of the venous system is one of the extraordinary phenomena pertaining to diseases of this organ; but in some circumstances, true beats, of more or less regularity, are observed in the veins near the heart (as the internal and external jugular). As this phenomenon constitutes a very valuable sign in cardiac diseases, it is important to distinguish the swelling of the veins from congestion, from the venous beats, or the swelling from regurgitation of these vessels. It is for the purpose of establishing this distinction, that M. Gendrin proposed a very ingenious process, which consists in making compression on the part of the jugular vein, most distant from the heart. If, in this case, the vessel remains empty and collapsed, the swelling of the veins does not depend on the regurgitation of the blood of the heart into the veins, but from an insufficient depletion of these vessels: if, on the contrary, after having forced the blood from below upwards into the external jugular vein, and placed a finger on the most inferior part of this vessel, the blood is seen to reascend in the vein, and there produce pulsations, it may be affirmed, that the swelling of the veins depends on the reflux of the blood which distends the heart. The swelling of the veins with beats does not belong, therefore, to congestion, but rather to regurgitation. These beats, appreciable only to the sight are perceived especially in the superficial veins of the neck; and sometimes in the internal jugular. They may be simple or double: when *simple*, they are always synchronous with the systole of the heart; when *double*, they are composed of an undulatory tremor which precedes the systole, and by a true beat, synchronous with the systole of the ventricles. The simple pulsations of the veins (*systolic*) may depend, either on a deficiency of the tricuspid valve, or on a considerable dilatation, with hypertrophy of the right ventricle; in this last case, the phenomenon is due to a regurgitation of the blood placed between two segments of the now thin and badly supported tricuspid valve, the motion of which is transmitted to the blood of the auricle and superior veins (Hope). The double pulsations depend, first, on hypertrophy of the right auricle; second, on deficiency of the tricuspid valve, or on dilatation with hypertrophy of the right ventricle. These double beats are at their maximum where there is contraction with deficiency of the right auriculo-ventricular orifice. In the mucous membranes, the congestion is not less evident; and hence we see it supervening in the intestinal tube, and characterized by a punctuated redness, or by flakes of mucus in the stomach and intestines. This congestion, which is always dangerous, is announced by anorexia, eructations, severe pains extending through the abdomen, and later by serous diarrhœa, sometimes very abundant, and which is often accompanied by very prominent hemorrhoidal tumours.

The functional vascular apparatus of the lungs (which is so immediately connected with the action of the heart), is in some measure the first to suffer from alteration of this organ. Hence it is in the lung that the first general phenomena make their appear-

ance when they are dependent on embarrassment of the venous circulation; the first of these phenomena is the œdema of the lungs, which is indicated by a dry cough, considerable dyspnœa, evident diminution of resonance at the base of the lung behind, and the presence at this point of a crepitant moist râle with distinct bubbles; and, anatomically, by an increase in the consistence of the pulmonary parenchyma, which is like paste, its pale and greyish colour, and in its general infiltration with a yellowish, frothy serosity. It is not uncommon to find, at a more advanced period in the diseases of the heart, the lung tumefied, with an increase of density in certain points, and infiltrated with a yellowish-red serosity, which flows with difficulty. These cases of œdema may be easily confounded with pneumonia; for independently of considerable dulness, there is found either a moist crepitant râle, or a true tubular sound. It is true, that the absence of the rusty sputa, of the symptomatic fever, and of the pain in the chest, prevent us from confounding these two diseases; but these cases are not less troublesome. The pulmonary œdema may manifest itself, and disappear many times; it often alternates with a phenomenon which we shall soon study under the name of colliquative diuresis, and has its maximum of intensity in the cardiac diseases, in which the circulation is found most embarrassed, as in the case of deficiency of the mitral and tricuspid valves. The pulmonary œdema uniformly precedes the hydropic effusions which take place in the serous cavities.

Pulmonary œdema precedes also that state of sanguineous engorgement, in which this organ takes on a reddish-brown colour, loses its elasticity, and furnishes after incision a considerable quantity of blood of a violet-brown colour. This state of congestion of the lung, which is generally accompanied by hemorrhage, is characterised by extreme dyspnœa, with partial dulness, and by the moist crepitant râle, as in œdema.

The liver, which is the centre of all the venous system of the vena porta, and which empties itself by large outlets into the vena cava inferior, suffers more than any other organ from obstacles which oppose the return of venous blood. This congestion of the hepatic organ is made known by considerable tumefaction. On one side, it extends inferiorly below the border of the false ribs on the right, and it descends in front to the level of the umbilicus; it forms a shining tumour, and most often yielding to pressure, which preserves entirely the form of the liver. In another direction, this organ ascends by its convex face, which is rendered evident by auscultation and percussion. The swelling of the liver adds often to the dyspnœa, and it announces almost always incipient ascites, especially when the inferior extremities are in a state of œdema, and there is meteorism of the abdomen. It is never complicated with jaundice, unless the liver be the seat of a phlegmasia: in severe cases, it is accompanied by meteorism, vomitings, diarrhœa, and swelling of the hemorrhoidal veins, with or without hemorrhage. These congestions of the liver are evidently caused

by those diseases in which the return of the venous blood is most impeded; thus, they are most often seen in diseases of the auriculo-ventricular orifices, and especially of the left. They are produced more rapidly in proportion as the obstacle is situated near the venous circulation: if it is in the right ventricle, the congestion will happen much sooner than if it was in the left ventricle. Hepatic congestions are most often accompanied with congestion of the lung, since it is through the medium of this organ that the venous circulation becomes arrested. We have already spoken of the congestions which take place in the intestines, and which succeed those of the liver. It is easy to comprehend how this congestion may take place in the spleen, and finally, by degrees, reach, not only all the organs which contribute to form the branches of the vena porta, but also all those which pour their blood into the inferior vena cava. The congestion of these last organs is indicated most commonly by a particular circumstance, noticed for the first time by M. Gendrin, and which consists in the habitual excretion of a quantity of urine, much more abundant than the amount of liquids taken in. It is generally during the night that thisexcretion is principally abundant; the urine is white, watery, and almost always without sediment; it contains no albumen; its quantity varies with the stage of the disease; but augments as the latter advances. The *colliquative diuresis* is not always constant: when it does exist, the dyspnœa is generally diminished. The diuresis almost always precedes the anasarca, and is caused by obstacles which are situated at the orifices, and particularly at the auriculo-ventricular ones. It is, however, observed in some cachexies, in chlorosis, for example.

The brain is not protected from the congestions which take place in the other organs; the connection which exists between the cerebral affections and the diseases of the central organ of the circulation has always been noticed. Congestion under these circumstances is announced by the following symptoms: dull and continued headache, weight in the head, inclination to sleep, injection of the capillaries of the face and conjunctiva; at a later period succeeds a comatose state, and the symptoms of a paralysis more or less extensive.

2. *Hemorrhages.* — There is only one step from congestion to hemorrhage. Thus, we see patients who, after having presented for a long time symptoms indicative of congestion of the brain, are suddenly attacked with all the symptoms which point out a cerebral hemorrhage, viz., sudden loss of consciousness, circumscribed paralysis, &c. These cerebral hemorrhages and the encephalic sanguineous congestion which precedes them, have been generally considered as the immediate effect of the excessive impulse given to the column of arterial blood by the left ventricle of the hypertrophied heart. We shall examine, under the article *Hypertrophy*, whether this opinion be well founded.

It is not only in the brain, but much oftener in the lungs, that

hemorrhages are observed; as we can readily understand by a knowlege of the situation of the lung. This organ, placed between the right and left side of the heart, receives continually, through the pulmonary artery, the blood which it is to transmit to the left auricle by means of the pulmonary veins; but if any material obstacle exists in one of the orifices of the left ventricle, and especially in the left auriculo-ventricular orifice, the blood which at first stagnates in the left auricle, accumulates gradually in the pulmonary veins which are distended, then in the delicate vessels of the lung. These last either allow the blood to transude on their surface (*hemorrhage by exhalation*), or else are torn to give passage to this liquid (*pulmonary apoplexy*). Of these two forms of hemorrhage, the most common is certainly the pulmonary apoplexy (*pneumo-hemorrhagy*). This affection, characterised anatomically by the sanguineous infiltration of a greater or less number of lobules, and by their conversion into a black, homogeneous tissue, is commonly announced by intense dyspnœa, more or less dull pain in the side, palpitations, expectoration of a brown, and even black blood, dulness on percussion, and by the moist crepitant râle more or less mixed with the tubular sound. In general, pneumo-hemorrhages only supervene in the first periods of the disease of the heart, when the organism is not much deteriorated, that is, in persons who have not had œdema, and who still present all the appearances of good health. At a later period, when the contractility of the heart is enfeebled, and the pulmonary vessels become by degrees habituated to this sanguineous congestion, these hemorrhages become rare.

Hemorrhages of the liver, and of the gastro-intestinal tube, ordinarily take place at a more advanced period in the diseases of the heart. These, when they are very abundant, often bring temporary relief for a certain length of time; finally, in the last stages of the diseases of the heart, there are sometimes observed sanguineous effusions, in the tissue of the eyelids, for example, and in the subcutaneous cellular tissue of the face. These sanguineous congestions may be increased in an alarming manner, and may even take place in the heart itself (Gendrin).

3. *Serous Infiltration.* — The serous infiltrations are seen sooner or later in almost all severe diseases of the heart. They first take place in the cellular tissue (*œdema, anasarca*), and later in the serous cavities (*dropsy*); commencing always in parts of circumscribed bodies, especially in the inferior extremities. At first the œdema only supervenes at the end of the day, and disappears on lying down; it induces no change of colour in the skin, and only a feeling of oppression: it preserves a long time the impression of the finger; but in proportion as the infiltration progresses and advances to the trunk, it no longer disappears completely on lying down. The œdema next occupies the cellular tissue of the parietes of the thoracic and abdominal cavities; and it may even become general. When it shows itself in the superior extremities, it is

especially the right which is the most affected. We have for a long time sought for the reason of this. May it not be owing to the habit which patients have of lying on the right side, and consequently rendering it lower?

To this general anasarca is added very often considerable dyspnœa, proceeding from the pulmonary œdema, which is a constant attendant, as well as from the infiltration of the thoracic parietes, which concurs to embarrass considerably the movements of the muscles. The anasarca is always accompanied by a considerable deterioration of the functions. The organs which only receive very poor blood fall into a state of inertia; the muscles become atrophied; the skin and all the mucous membranes become dry; the renal secretion diminishes; so that, when we have succeeded in causing the anasarca to disappear, we always find the strength of the patient so enfeebled, and the general complications so aggravated, that there is little hope of re-establishing the healthy organism. It is only when the anasarca has increased to a very great extent, that infiltrations take place in the serous cavities; they are at first effused into the peritoneal cavity, then into that of the pleura and pericardium, and sometimes even into that of the arachnoid.

These serous effusions add to the gravity of the primary disease: the ascites renders the respiration more difficult, increases the embarrassment of the circulation in the abdominal vessels, induces vomiting and constipation; the hydrothorax diminishes the extent of the pulmonary surface, contracts or diminishes the space for hematosis, and increases considerably the dyspnœa; the effusion into the pericardium augments the irregularity in the movements of the heart; and the serous effusion into the arachnoid, which throws the patient in a state of profound coma, often puts an end to his agonising sufferings.

4. *Gangrene of the Extremities.* — Gangrene of the extremities is sometimes seen in individuals of an advanced age, as a complication of the diseases of the heart. It only happens at a very advanced stage of the disease, when the whole organism is much deteriorated; that is, when the anasarca is constantly reproduced, and when the patient is much emaciated and complexion deteriorated; it most often happens after scarifications of the inferior extremities; the presence of œdema is always one of the principal causes. Sphacelus commences ordinarily in the skin of the toes or fingers, which are covered with livid spots, or in the lips of the scarifications, which become black; at the same time the limb is the seat of acute pains, with a feeling of coldness; the spots increase in extent; phlyctenæ are formed, and the sensibility is extinguished in the points where the epidermis is raised. These spots rapidly ascend to the trunk in increased numbers, and below them the skin is always mortified. In proportion as the disease extends superiorly, the pulsations of the artery in the limb affected cease to be perceived, and the arterial tube is converted into a cord filled

with coagulated blood. The veins of the limb become nearly effaced, and are only recognised by black lines which furrow the surface of the limb: at the same time, the beats of the heart grow feebler; then follows adynamia, wild delirium, eschars on the sacrum and great trochanters; finally, the patient dies comatose seven or eight days after the invasion of the sphacelus. Such is the gangrene which is a consequence of diseases of the heart. Gangrene of the extremities may also be caused by diseases of the arterial tubes: it is this species that the surgeons have described under the name of *senile gangrene;* but this is not the place to speak of it.

5. *Dyspnœa.* — The serous and sanguineous congestions which almost always have their seat in the lungs, and the difficulty of hematosis, which is a consequence of them, produce always some difficulty in the respiratory functions. The patients do not at first take notice of their dyspnœa; afterwards they find that they are much out of breath when they take exercise. It is not rare to find, in the working classes and in children, individuals who present the physical signs of severe diseases of the heart, and yet say that they have never felt dyspnœa. The reason is, that these individuals pay little attention to their sensations, and that, as the dyspnœa comes on gradually, they become accustomed to it, and never think that they are afflicted with any disease.

The dyspnœa increases continually, and often imposes on the patients the necessity of preserving the most absolute repose. This affection is not always felt to the same degree. When at rest, the patients suffer little or none; but deviations in diet, moral emotions, and corporeal labour increase its intensity. It has not always the same causes: sometimes, it is due to the sanguineous or serous congestion of the bronchial mucous membrane; sometimes to a chronic bronchitis, or to a very marked pulmonary emphysema. But the fixed cause of this dyspnœa is always found in the insufficient oxygenation of the blood; either because the air only penetrates the aërial vesicles and bronchial ramifications in a very small quantity; or because the permeable state of the mucous membranes prevents the blood from being sufficiently exposed to the action of the air; or, finally, because the blood does not flow in sufficient quantity into the pulmonary vessels.

Dyspnœa is generally one of the first signs of diseases of the heart: sometimes it is habitual, and sometimes it is paroxysmal. Most commonly habitual, it also presents exacerbations. It is this last which constitutes one of the great varieties of asthma, that is called *cardiac asthma.*

Cardiac asthma presents various characters; sometimes it is *humid,* for example, when the lungs are in a permanent state of engorgement, as happens in the case of contraction of the left auriculo-ventricular orifice; it is *dry* when the engorgement of the lung is only temporary, as happens in simple hypertrophy; finally, it may be *convulsive.*

Sometimes the attacks of asthma come on suddenly; sometimes (in severe cases, for example), they are announced by some precursory troubles, as a feeling of weight in the epigastric region, distension of the abdomen, pain, oppression, constriction in the forehead with some beating, and an indefinable sensation of oppression and anxiety in the præcordial region. At other times, the invalid is uneasy, irritable, morose, and it may be apathetic or melancholy. Whatever was his previous state, the attack begins in the following manner: the patient, if he is lying down, wakes up suddenly with violent palpitations, with an agonizing sensation of oppression in the præcordial region, and a considerable tightness in the chest, as if it was forced in a vice: he raises himself upon his seat and demands instantly air: the respiration is noisy, and is accompanied with violent efforts of all the respiratory muscles; the inspirations are long, the expirations short and imperfect; the whole external surface feels cold; the face is pale or livid; the pulse frequent, small, feeble, often irregular and intermittent. In proportion as the attack loses its intensity, the oppression and tightness of the chest diminish, the respiration becomes less frequent, less loud, and less laboured; the pulse slower, fuller, and more regular; the patient retains only a slight constriction of the chest, and a slightly noisy respiration. Finally, the attack terminates by a very abundant expectoration of a viscid liquid, which is transparent, or by a considerable diaphoresis; sometimes by a copious evacuation of pale and clear urine. As long as these evacuations, which constitute the crises of the attack of cardiac asthma, and which announce the disengorgement of the pulmonary system, do not take place, it is to be feared that the attack will soon reappear. In general, the attacks of asthma are more complete if the obstacle to the circulation is great, and the pulmonary congestion continues long. Thus, in the case of disease of the valves with considerable dilatation, the attacks often last for five or six hours. In proportion as the organic disease of the heart progresses, the attacks of asthma, which at first only returned at distant intervals, come nearer to each other; the respiration, always short, is now quick and laborious from the least exercise, or from the least moral emotion; finally, in the last period of this disease, the cardiac asthma is persistent. Unable to remain in the horizontal position, the patients remain for weeks and whole months in almost a vertical position, and sustained by pillows, their body bent forward, their elbows placed on their knees, their eyes widely open and staring; their eyebrows raised, and their nostrils dilated. Their features are dark and haggard, and their head is drawn backwards at each inspiration; they cast around them a mingled look of horror and entreaty; sometimes praying with an almost inaudible voice, and with plaintive cries, or in feeling and broken accents, for the assistance which has been freely administered to them in vain: sometimes accusing the impotency of medicine; sometimes, finally, in the agony of despair, letting their head fall on their chest, and murmuring a fervent request that

death would put an end to their sufferings. For some hours, sometimes only for some minutes, they experience a delightful interval of calmness. They flatter themselves with the hope that their distress is passed, and their cure is at hand; but this hope soon vanishes; the short sleep which they enjoy is interrupted by the horrors of an unfortunate dream; they raise themselves up, uttering piteous cries. Finally, the respiratory muscles, overcome by the efforts that the instinct of self-preservation has imposed upon them for so long a time, participate in the general exhaustion, and refuse to fulfil their functions. Then the patients expire in the midst of a last inspiration (which is most rarely the case), or fall exhausted on their bed; the respiration in this case is short, raucous, almost absolutely diaphragmatic; the skin becomes violet, the pulse almost imperceptible, and the patients fall into a state of comatose asphyxia. This comatose state may last some hours; we have seen it last for three days.

6. *Nervous Disorders.* — We include under this head all those disorders of innervation which have an influence either over the general or special sensibility of certain organs, and tissues. Amongst these disorders, we notice especially acute pains which the patient experiences in the epigastric region when he partakes of the indulgences of the table; and which are accompanied by eructation in all cases. We must also notice those pains which radiate from the epigastric region towards the left shoulder, and which are accompanied by more or less painful numbness in the superior extremity of the same side (*angina pectoris*). These pains may not at first descend below the deltoid muscle, but they soon extend along the internal part of the arm to the elbow, sometimes even to the extremities of the fingers, following the course of the cubital nerve. Their duration is almost always short: when they are prolonged, they give rise to a suffocating dyspnœa, to violent palpitations, to syncope, and even to convulsions. Generally the attack is induced by all the causes which excite the action of the heart, as walking, running, the pleasures of the table; it seldom lasts more than ten minutes, and still more rarely continues for half an hour or an hour: in this last case the danger is imminent. *Angina pectoris* is only present in very advanced and severe disease of the heart. It is at its maximum in cases where there are osseous or cartilaginous and steatomatous degenerations of the heart and large vessels, or where there is hypertrophy and considerable dilatation of the organ with or without softening of its parietes.

Such are the principal general symptoms which, joined to the local ones already mentioned, may serve as elements in the diagnosis of diseases of the heart. But it is not sufficient to be acquainted with these phenomena, and their relation to their causes; we must also, in order to insure a correct diagnosis, determine the modifications of cardiac diseases at their different periods, otherwise called the order of succession of the different lesions of the heart and large vessels. These changes succeed each other in a particular

order. To give an idea of this, reserving ourselves for a fuller investigation hereafter, we cannot do better than introduce a passage from the work of M. Gendrin on the Diseases of the Heart. "An inflammation of the aorta, or of a great artery, is developed, and extends by degrees to the whole arterial system; next in order to the arterial phlegmasia and to the alterations which it causes in the vascular coats, palpitations supervene. In consequence of the palpitations, which are only marks of the increased action of the heart, this organ, or at least its left ventricle, is hypertrophied; and the arterial circulation is found in all conditions except those of health. The hypertrophy increases, and at the same time the heart dilates. At a still later period, notwithstanding the hypertrophy of its walls, in consequence of the dilatation of its left ventricle, the action of the heart on the blood diminishes. •At a period still more advanced, the aortic valves become indurated and disappear; and this disappearance, favoured by the hypertrophy of the heart, induces a new modification, which changes again the form of the disease. The arterial system, forced to continue the circulatory impulsion by itself, becomes hypertrophied; whilst this arterial impulsion becomes more and more feeble in the diseased parts of the vascular apparatus. These disorders continuing, and increasing daily in the circulatory apparatus, all the organic functions become changed and enfeebled; the skin is dry, the urine watery, the patient falls into a state of leanness and cachexia, which transfers the influence of the disease to the whole organism. In the interior, the liver and the abdominal vascular organs, gorged with blood, increase in volume; the pulmonary system is loaded; the heart, dilated subsequently, does not allow the portions of the mitral valves to approach; the blood flows back into the lungs, and all the phenomena which indicate embarrassment in the circulation of these organs, such as pulmonary infiltration and hemorrhages, appear."

Mode of Investigation in the Diseases of the Heart.

One of the causes which has most retarded the progress of the pathology of the heart, is certainly the want of method in the investigation of the morbid changes of this organ. The diseases of the heart are composed of a number of phenomena, all having a particular signification. It is necessary, therefore, to proceed to their examination in a methodical manner, in order that none of them may be omitted. This is the mode which appears to us most proper; it is similar to that which M. Gendrin has followed.

To examine properly patients affected with diseases of the heart, it is necessary to place them in an almost horizontal position; to take away all clothing that may either embarrass the motions of the chest, or produce artificial sounds by friction. It is preferable to examine them in the morning, after sleep. It is necessary to

uncover all the præcordial region in men, and place only very fine linen on the anterior surface of the chest, in women.

We must first find out, by the sight and touch, that there is on anomalous tumour seated in the præcordial region, and that the thoracic cavity presents no kind of deformity; then, applying the hand over the præcordial region, we must try to find exactly the seat of the apex of the heart, and its distance from the axis of the sternum. At the same time we must judge, by the hand applied over the heart, of the force, the extent, the number, the duration, and the regularity of the beats or impulse of this organ. A knowledge of the position of the apex of the heart is one of the most important elements in diagnosis, since it permits us to determine the length of the organ, by measuring the distance which separates this point of the thorax from the intercostal space where we have said that the base of the heart is invariably fixed.

We then make percussion over the præcordial region, by striking lightly, with one or two fingers of the right hand on one of the left hand, applied immediately over the intercostal spaces.

We thus estimate the extent and degree of dulness, and at the same time also the position of the anterior border of the lungs, the volume of the heart, and that of the pericardium.

We pass, next, to the auscultation of the præcordial region. In this examination, the ear is most often made use of; although, when we wish to limit the surface to be auscultated, or when the structure of the parts opposes the immediate application of the ear, the stethoscope is used. The form of this instrument is not of much consequence, provided that its ligneous fibres be continuous from one extremity of the cylinder to the other. The rules to follow for the application of the stethoscope in the diseases of the heart, are the same as those for diseases of the chest; it is only necessary to take out the top, which Laennec added to it. The first thing to do in the auscultation of the heart, is to isolate it as much as possible from the sounds which take place in the chest. When these are very intense, it is necessary to tell the patient to hold his breath for a few minutes: applying then the ear over the place where the apex of the heart is seated, we hear sounds, and can determine the modifications which they experience in their force, extent, tone duration, and rhythm, as well as the modifying anomalous sounds, by referring them to the two functional acts and to the two sounds of the heart. From the apex of this organ we extend the examination to the left, at first transversely, then ascending to the border of the pectoralis major, and descending to the inferior limits of the left hypochondrium (as it is in this space that we find the maximum of the sounds which are produced in the left ventricle), in order to examine whether the normal or abnormal sounds increase or diminish in going towards this point, whether any anomalous sounds are added, taking into account the respiratory phenomena, and the transmission of sounds which may take place in consequence of a disease of the left lung. We then go to the

right, and within the space from the apex to the sternum (maximum point of the sounds which take place in the right ventricle), examine the modifications in the phenomena which are perceived at the apex. In this last examination, we must take care, and not be misled by one cause of error. The anomalous sounds of the aorta are often perceived at this point, and may be mistaken for anomalous cardiac sounds. To avoid this error, it is sufficient to remark — 1st. Their constantly synchronous character with the systole of the heart, which belongs to the aortic murmurs. 2d. Their extension over the whole course of the sternum, often even under the linea alba; whilst the murmurs of the right side of the heart may be systolic or diastolic, and never are prolonged to this extent.

Setting out again from the apex of the heart, we ascend from below upwards, over the course of this organ to its base. In our progress, the modifications which the normal sounds undergo, and the changes taking place in the anomalous ones as heard at the apex, are rendered evident. We make the same examination when we have reached the base, and we note in the course of the pulmonary artery, then of that of the aorta and its ramifications, what modifications the normal or abnormal sounds undergo; and whether new ones are developed. We follow these sounds, particularly in the brachio-cephalic trunk, the subclavian, the external primitive carotid, sometimes the internal, by applying the stethoscope on the base of the orbit, as M. Gendrin does. We then follow the course of the aorta in the abdomen, and of its iliac and crural branches.

The investigations to be made respecting the arterial circulation demand much care. Thus, when we examine the arteries which are enclosed in the thoracic cavities, we must, as much as possible, isolate the sounds of these vessels from the respiratory sounds, and especially from the normal and abnormal ones of the heart. In the abdomen, we must also distinguish the arterial sounds, 1st, from those of the heart, which latter sometimes extend even to the abdomen; 2d, from intestinal borborygmi; 3d, from the muscular sound which is developed in the abdominal parietes when they are not sufficiently relaxed, and when they contract under the pressure of the stethoscope. When, on the contrary, we examine arteries which are only covered by soft parts, there are some especial precautions to take. We must not press too much with the stethoscope, nor relax the parts which cover the artery. When we auscult the arteries of the neck, the patient should have his head supported by a pillow, and a little stretched, but the face turned forwards. Next we must examine by making the parts tense: in the neck we turn the head to the side opposite to that on which we examine, raise the chin, &c., to ascertain whether the modifications of the arterial sounds are similar on both sides. At the same time, we recognise by the touch the state of these arteries; we study the effects of pressure on them, the modifications of the beats, in their force, frequency, and rhythm. Resuming the exploration of

the trunk behind, we ascertain, over the course of the lateral parts of the spine, whether the sounds of the heart and great vessels are heard in these points, and what are the modifications which they have undergone.

Finally, we conclude by noting the colour of the face, the state of the cutaneous capillaries, and of the different mucous apertures, the state of the pulse in different parts of the body, the condition of the viscera; at first of the lungs, then of the abdominal contents: we carefully note, also, the state of the abdomen, the presence of infiltrations, or serous effusions, &c.

CLASSIFICATION.

We shall divide the diseases of the heart intothree classes:

1st. Inflammatory Diseases.

2d. Organic Diseases.

3d. Nervous Diseases.

This classification is open to criticism, because it ranks among the organic diseases those which most resemble inflammations: but, as a good classification would rigorously infer a knowledge of the intimate nature of the diseases of the heart, at which we have not yet arrived; as the classifications can only be artificial means, whose end is to aid us in the study of numerous objects; and as, finally, all classifications of diseases have their faults, we request for this the indulgence that is given to all others.

FIRST CLASS.

INFLAMMATORY DISEASES OF THE HEART AND GREAT VESSELS.

CHAPTER I.

PERICARDITIS.

The name of pericarditis is given to inflammation of the external membrane of the heart. It may be acute or chronic.

Anatomical Characters. — 1. *Alterations of the Pericardium.* — Redness is one of the appearances in pericarditis: sometimes it occupies the serous membranes; sometimes (and most commonly) it has its seat in the subjacent cellular tissue; it rarely is present in the whole extent of the pericardium, and is ordinarily presented under the form of more or less considerable spots, vascular arbori-

zations, or dotted lines. In some circumstances, where, if we judge from the thickness of the false membranes, the inflammation has been very active, we find no trace of redness. It is probable that, in these cases, the redness disappeared after death, as is often seen in inflammations of serous membranes. The pericardium loses, at the same time, its transparency and consistence: it appears almost always thicker than usual, although this effect is more often produced by an opaque false membrane, intimately adherent to the serous one, or by a thickening of the subjacent cellular tissue, than by that of this membrane itself. Its internal surface, when there is no effusion, is a little rough to the touch; at other times it is dry, shining, and, as it were, scaly; when there is effusion, it is lined by false membranes. Its external surface, which answers to the muscular tissue, rests most often on the softened cellular tissue; consequently the serous membrane is detached with the greatest facility.

2. *Products of Secretion.* — The pericardium when inflamed secretes from the same vessels both serosity and lymph; the plastic lymph which is secreted separates by degrees from the serosity, spreads itself out on the folds of the pericardium, and constitutes false membranes. A small quantity of plastic lymph remains, however, generally suspended in the serosity, under the form of flocks or threads. The *false membranes* of the pericardium, when recent, are soft and tender; in progress of time they become more firm and consistent; most generally forming a continuous surface, which sometimes covers a portion and sometimes the whole of the pericardial sac. They are also seen in the form of masses or flakes; and give to the surface of this membrane, a rough, papular, or granular appearance: their thickness is commonly from one to three lines, but it may extend to an inch: their adherent surface is polished: their free surface is rough and variously figured; the latter may be covered with small depressions, disposed with some degree of regularity, and present the aspect of a finely reticulated or spongy tissue. When the false membranes are thick, their free surface is divided into a geater or less number of deeper cells or depressions, often of the diameter of a pea, and separated by marked partitions. These latter are sometimes irregular, stronger and thicker in one point than in another. We may compare this arrangement to that which is observed, when we have just separated two flat plates, which had been applied to each other after their having been covered with soft butter. When the partitions are more regular, the false membranes resemble (as Corvisart has remarked) the internal membrane or second stomach of a calf: when they are thick and rounded, they resemble perfectly a mass of small worms: very often they are hairy and in flakes, terminated by fringes like tow; finally, in some still more rare cases, they may be disposed in the form of transverse wavy wrinkles, so as to imitate the undulations of the sand on the sea-shore. This particular disposition of the free surface of the false membranes is due to

the continual movements by which the heart is agitated, or rather, as M. Bouillaud says, to the sudden separation and renewal of the two surfaces covered with a soft substance. The colour of the false membranes, which is at first a pale yellow, acquires by time a darker tint, which varies from cinnamon to a deep reddish-brown or mahogany colour; in this case they habitually secrete a sanguinolent fluid (*hemorrhagic pericarditis* of Laennec). Their organisation proceeds with great rapidity; it is a sort of reparatory effort, whose result is to create between the two folds of the pericardium true adhesions. When the quantity of liquid effused is much diminished, the pseudo-membranous laminæ which clothe the two opposite folds of the pericardium approach each other, touch and gradually unite. This union takes place through the medium of vessels which are transmitted successively into the states of spots, irregular lines, and finally, into an uniform vascular pinkness. When this pinkness disappears in its turn, the false membranes are converted into a perfect cellular tissue, which unites more or less intimately the two folds; and their organisation may be considered as complete. When the adhesion is recent, the false membrane is sufficiently thick and soft to be separated into two parts, each of which adheres to a fold of the pericardium; but when the disease is further advanced, the false membrane is more thin and resisting; and when the disease is of many years date, this false membrane only consists of a very thin bed of dense cellular tissue. Finally, the two folds may be completely united with nothing intermediate. In some cases of a more severe nature, the formation of the adhesions is not regular, and new membranes are disposed between the first false ones. The adhesions may be general or partial, when the inflammation has been partial, or when the deposit of lymph has only taken place at certain points. The partial adhesions, continually drawn out by the movements of the heart, become lengthened and converted into long laminæ or strings. In some fortunate cases, as M. Gendrin has shown, the general or partial adhesions may disappear, after a certain time, under the influence of absorption, and leave no trace of the disease.

After an attack of pericarditis, we find small, round, and soft granulations, or white or milky spots on the surface of the heart; these may be two or three inches in diameter and of the thickness of the nail. They are formed, in most cases, by a pseudo-membranous lamina, which exhibits all the characters of condensed cellular tissue, and beneath which the serous lining of the pericardium is found injected, but not thickened; at other times, they are constituted by hypertrophy of the sub-serous cellular tissue; they occupy principally the anterior face of the heart and large vessels which arise from it, and are capable of undergoing a variety of transformations, and of passing into the fibrous, cartilaginous, and even osseous state.

The *liquid effused* into the cavity of the pericardium is sometimes

transparent, and of a pale yellow colour, bordering upon green, or rather of a light fawn colour; more generally it is thick and cloudy, in consequence of membranous flakes which remain suspended in it; sometimes, also, the presence of a small quantity of true pus imparts a certain degree of milky opacity. In some very rare cases, true creamy pus, of a yellowish-green colour, is found without false membranes; lastly, the liquid may contain a greater or less quantity of blood. The quantity of the liquid is variable. Generally abundant during the period of the increase of the disease, it amounts often to a *litre** and a half, and even beyond this (four pints, according to Corvisart and Louis). It is rapidly diminished by absorption; and some authors even admit, that in certain cases where the inflammation is very acute, the absorption is so complete that you only find false membranes without serosity; others go still further, and admit the existence of pericarditis where there is no liquid effused — *dry pericarditis.*

We confess that we are much opposed to the admission of these cases of *dry pericarditis;* for, although the friction sound is recognised in the early periods of the disease, yet still, we are not disposed to acknowledge this class; since this friction sound is produced, even when there is liquid, provided it be in small quantity.

3. *State of the Heart.* — The muscular texture of the heart is generally unaffected; and may sometimes be redder or paler, more brown or yellow, more dense or fragile, than in the normal state. The internal membrane often participates in the inflammation of the pericardium. We refer, for the anatomical characters of this inflammation, to the chapter on *Endocarditis.*

Chronic pericarditis does not present any anatomical changes which differ materially from those that are observed in the advanced stages of acute pericarditis. The inflammation occupies the whole surface of the pericardium; the redness is of a deep and dark tint; there is more or less effusion, of a thick, flocculent, milky liquid, sometimes quite puriform, at other times, of a viscous, pultaceous, sanguinolent consistence. False membranes are often wanting, or only consist of thin and soft layers, which seem worn away by suppuration: at other times, the quantity of lymph is enormous, and the heart is in some degree oppressed by their weight. These false membranes may be the seat of numerous degenerations (tubercular, cancerous, &c.). In chronic pericarditis, the heart is often found hypertrophied; in some more rare cases it is atrophied.

Causes. — The causes of pericarditis may be divided into—1, *Physical causes;* these are sources of irritation directly applied to this membrane, as falls, blows, wounds, kicks, pressure on the præcordial region, the penetration of foreign bodies into the pericardium.

* Litre is equal to 2·1135 pints.

2. *Physiological Causes.* — To this head are referrible all those causes which give too great activity to the organs engaged in hematosis. If pericarditis be often met with in young persons, it is because with them the circulatory organs enjoy greater activity: the same cause will account for its presence in plethoric subjects.

3. *Pathological Causes.* — Among these, we place, as first in importance, the sudden suppression of cutaneous perspiration, hemorrhage, or cutaneous affection; purulent absorption; the propagation of a phlegmasia of the pleura or lung to the pericardium; the prior existence of an inflammation of the muscular substance of the heart, or of the endocardium; organic lesions of this apparatus; and above all, acute articular rheumatism. This last cause, whose influence had been already noticed in England by Baillie, and, later still, by Willis, Odier, Elliotson, &c., has been especially made known by M. Bouillaud in France, who has shown that, in more than half the cases of acute articular rheumatism, there is a curious coincidence of inflammation both of the pericardium and endocardium. The older physicians attributed this occurrence to a metastasis; but as inflammation of the pericardium comes on at the same time with the articular affection, and sometimes even precedes it, we cannot possibly refer it to this cause, and it would be better to attribute this transfer to what Bichat has called the *affinity of tissue.*

Symptoms of Pericarditis. — It is only of late that we have acquired an exact knowledge of this disease. Avenbrugger had already shown what advantage would accrue to diagnosis from the knowledge of percussion; but, the quite recent application of auscultation to the study of pericarditis, made by MM. Collin, Latham, Louis, &c., has thrown new light on this subject.

A. *General Symptoms.* — These are high *febrile reaction* preceded by general coldness, with a chill and shivering; a dry *cough*, returning in paroxysms, with little or no expectoration; hurried *respiration*, *dyspnœa* almost amounting to suffocation; extreme *anxiety* with jactitation, and sometimes with syncope; pulse frequent, hard, abrupt, sudden, full, and even jerking at first, but regular, and subsequently, under certain circumstances, feeble, intermittent, with unequal intervals, and irregular; it sometimes presents this character from the beginning. The *features* are drawn up and contracted, sometimes accompanied with a sardonic smile; there is *swelling* and *lividity* of the face; *infiltration* of the extremities; disorders of the nervous system, as delirium, which is ordinarily slight, and sometimes general convulsions; disturbance in the digestive apparatus, such as loss of appetite, greater or less thirst, and in some cases repeated vomitings.

B. *Local Symptoms.* — 1. *Local Physiological Symptoms.* These are — *a. Pain* in the region of the heart, of a pungent, burning, and lancinating character, radiating to the left shoulder and arm of the same side, increased by strong inspirations, by percussion, and by pressure made either on the præcordial or epigastric region;

this pain is more or less dull in some cases; it may consist of only a simple feeling of uneasiness, or be entirely wanting; but in this last case, pressure made on the præcordial region, or on the epigastrium, always causes pain. When the pain is acute, it forces the patient to lie on the left side, and prevents him from assuming the horizontal position. *b. Various modifications in the beats of the heart:* The beats are stronger and more frequent than in the normal state, sometimes regular; at other times irregular, unequal and intermittent (palpitations). When there is little or no effusion into the pericardium, the *impulse* of the heart preserves its sudden and vigorous character; if the effusion is considerable, the impulse becomes feeble, tremulous, irregular, and unequal; it is, in a measure, undulating, and does not coincide exactly with the first sound. *c.* A projection or anomalous *prominence* in the præcordial region, which is met with in some cases.

2. *Local Physical Symptoms, or Symptoms furnished by Percussion and Auscultation. — Percussion.* In pericarditis the dulness in the præcordial region increases in a direct proportion to the quantity of liquid effused; it becomes easily appreciable when there is half a litre of liquid; it commences below, and at first extends transversely; it then ascends to the level of the second rib, when the pericardium is very much distended. Authors tell us, that by changing the position of the patient we can vary the level of this liquid, but we have never succeeded in doing this.

Auscultation. — When called to a patient affected with pericarditis, if it be only a few hours after the development of the disease, and when but a very small quantity of liquid is effused, we perceive one of those sounds which have been described under the name of *friction sounds;* but in proportion as the effusion increases, the first sound of the heart and the murmurs, if they exist, which are seated at the auriculo-ventricular orifices, are more obscure than in the normal state, because they are heard through a mass of liquid and lymph.

The second sound is always perceived almost as distinctly as in the healthy state, by our following the course of the large vessels. The pericardiac friction sounds, which constitute one of the most important signs of pericarditis, result from the friction of the opposed rough surfaces of the pericardium; and, consequently, they can only exist when there is a very small quantity of liquid effused into the sac. It is generally admitted that these sounds are met with in three principal circumstances — 1st, at the beginning of pericarditis, before the occurrence of effusion; 2d, in those cases which bear the name of *dry* pericarditis, that is, without the effusion of liquid; 3d, at a more advanced stage of the disease, when the absorption of the effused liquid permits the rough surfaces to come in contact. The friction sound is almost always double; it accompanies the two sounds of the heart, and coincides with the movements of this organ in the pericardium. Sometimes, however, it is stronger with the first sound, and in

some rare cases it is limited to this sound exclusively: it rarely passes beyond the limits of the præcordial region, and never extends along the course of the great vessels; it presents, as regards pitch, numerous varietes, which we have already spoken of, and which appear to bear a relation to the firmness and rugosity of the false membranes, the quantity of liquid effused, and the greater or less intensity of the impulses of the heart. These friction sounds can only be confounded with the valvular murmurs which take place in the interior of the heart. But we shall review the differential diagnosis of the valvular murmurs and friction sounds of the pericardium, at the same time that we establish the differential diagnosis of endocarditis.

Symptoms of Chronic Pericarditis. — General Symptoms. When an attack of acute pericarditis is not resolved in an interval of from ten to fifteen days, it is said to pass into the chronic state; it is also called chronic when, from the beginning, it attacks slowly and insidiously, and the symptoms are not well-marked nor violent. The general symptoms which announce a chronic pericarditis much resemble those incident to the acute disease; but they have less intensity; the fever, instead of being high and inflammatory, puts on the characters of the hectic, and at different times has marked exacerbations. The anxiety and sleeplessness are less; the position of the patient is less constrained; but the face always presents a cachectic tint; and the inferior extremities are often œdematous.

Local Symptoms. — 1. *Physiological Symptoms.* The præcordial region is constantly the seat of a feeling of fulness and heaviness; the beats of the heart and the impulse are more feeble than in acute pericarditis, unless there be adhesions of the pericardium, or hypertrophy: sometimes there are neither intermissions nor irregularities. The arching of the præcordial region is often observed in chronic pericarditis, especially when it has induced hypertrophy of the heart.

2. *Physical Symptoms.* — They are the same as those of acute pericarditis; that is, augmentation of dulness on percussion, obscurity of the sounds of the heart, and friction sounds, when there is little or no liquid effused.

Diagnosis. — When the pain is seated in the præcordial region, and is aggravated by pressure; when there is increased action of the heart, and a violent febrile reaction, the existence of pericarditis may be suspected. If, besides, the pulse is feeble, trembling, irregular, &c., without our being able to detect the cause; if we observe all the signs of embarrassment in the circulation: and finally, if there is dulness on percussion, and obscurity in the sounds of the heart, there can be no doubt of the existence of pericarditis. But it may so happen, that there is neither pain nor considerable change in the pulse, no difficulty of respiration, no dulness appreciable by percussion, nor feebleness of the sounds of the heart. If, however, the beatings of the heart be violent and bounding without any manifest

cause; if there be anxiety and a high febrile reaction; and finally, if we recognise the friction sound of the pericardium, there is little danger of our being mistaken in diagnosticating pericarditis. As we have already seen, the diagnosis of acute pericarditis, in which we are assisted not only by general symptoms, but also by the results of percussion and auscultation, presents great precision. The most difficult cases are those complicated with diseases of the brain and other organs, and which divert the attention that ought to be given to the examination of the heart. We may say plainly; if it was a general custom to place the hand over the præcordial region, as often as it is placed on the pulse in all inflammatory diseases; if the heart was examined with as much care by percussion and auscultation as the lungs are, it would be a rare occurrence to mistake a pericarditis. This pretended diversity and variability of the symptoms, in which most of the pathologists now place the cause of obscurity of pericarditis, are still not without value in throwing light on the disease. Although they may cause temporary hesitation in the mind of the physician, yet they furnish valuable indications. These multiplied variations are in a direct proportion to the nature and progress of the anatomical changes of structure, and consequently to the progress and the stage of the disease. Generally, it is in the difference of the quality and quantity of the effusion that we must seek the difference in the aspect of the symptoms. If the effusion consists principally of plastic lymph, if the serosity has been rapidly absorbed, in a word, if the adhesions of the pericardium are rapidly formed, the movements of the heart present, during the whole period of the disease, the same vigour and regularity as at the beginning: the pulse retains its characteristic force, hardness, and regularity; the patient is less constrained in his postures, and life may continue for several weeks, even though the inflammation has not been removed. This mild form of the disease is observed still better when, instead of producing adhesions of the pericardium, it rapidly progresses to the state of resolution. If, on the contrary, the serous effusion be not absorbed, the movements of the heart are impeded by the mechanical compression of the liquid on this organ; and hence ensue a small, intermitting pulse, and all the symptoms which announce embarrassment of the cardiac circulation, such as feebleness, dyspnœa, anxiety, coldness of the extremities, lividity of the integuments, &c. If the serous effusion be abundant at the beginning, all these symptoms manifest themselves early in the disease; but as, in general, two or three days are required for a considerable accumulation of the liquid, to those which we have just described, suddenly succeed the symptoms indicating a strong and regular action of the heart. It must, however, be confessed, that there are certain cases of pericarditis, in which all the symptoms of difficulty of the circulation are observed, and there is still very little effusion of liquid. In most of these, there are very thick false membranes which act on the heart, in the same manner as the effused liquid: in other

cases, it is impossible to explain the occurrence of the accidents otherwise than by supposing complications of other cardiac affections (carditis, endocarditis, polypi of the heart), or of the pulmonary organs (pleurisy or pneumonia), or by an increase of irritability. According to M. Bouillaud, the great differences observable among the general symptoms of pericarditis depend rather on a violent pleuritic or pleuro-pneumonic complication, than on the pericarditis itself. No doubt these complications aggravate the symptoms, and impart to them the highest degree of intensity; but this opinion of M. Bouillaud cannot be admitted in an absolute manner, since we often see pericarditis with dangerous symptoms, without any trace of complication. We have now considered the general diagnosis of pericarditis; there yet remains to be noticed the differential diagnosis.

Pericarditis can only be confounded with inflammation of some one of the thoracic viscera, and especially of the pleura. These complications which, from the time of Corvisart, were one of the greatest obstacles to the diagnosis of pericarditis, no longer embarrass us, since the introduction of auscultation. It is well known that pleurisy, independently of the general signs, is recognised by the following physical ones: dulness on percussion, commencing at first in the posterior and inferior part of the chest, and only extending forwards at a later period in the disease; diminution or complete absence of the vesicular murmur in all the points which are the seat of the dulness; bronchial respiration, and œgophony, when there is only a small quantity of liquid. Pneumonia, besides these general ones, and in particular rusty and viscid sputa, is announced by the following signs: in the first stage, crepitant rhonchus, and commencing dulness on percussion; in the second stage, cessation of the crepitant rhonchus, and of the respiratory murmur; dulness evident on percussion; tubular sound and bronchophony; slight dilatation of the affected side, increase of the vibratory tremor of the voice. In fine, bronchitis is recognised by the presence of mucous, sibilant, and sonorous rales, and by the absence of anomalous dulness. If no one of these sounds be found, we are forced to the conclusion that the heart is the seat of the disease; but if, on the contrary, some of them are recognised, they should be compared with those which properly belong to the inflammations of the pulmonary organs, taking also into careful consideration the anomalous sounds which supervene in the heart, as well as the direction in which the dulness is perceived. After all, if we are in doubt, it would be proper to act as if this inflammation existed, adapting, at the same time, the treatment to the pulmonary complications as they arise; because it is much easier to subdue the diseases of the heart at their commencement, than when they have long had hold of this organ. There remains the differential diagnosis of endocarditis, which will be considered in the following chapter.

The obscurity of the symptoms of chronic pericarditis, and espe-

cially of this disease at its commencement, renders its diagnosis more difficult than that of acute pericarditis. Notwithstanding, with the aid of auscultation, these difficulties in a great measure disappear. In short, if a patient, who had not had previously disease of the heart, presents all the symptoms of this disease, with general emaciation and hectic fever; if he refers the attack of the disease to a blow or fall on the chest, to acute articular rheumatism, or to an inflammation with pain in the præcordial region, we may suspect chronic pericarditis; if to all these symptoms be added dulness on percussion, or the pericardiac friction sound, no doubt can exist respecting the existence of this disease.

Forms, progress, duration. — We think that two forms of pericarditis may be recognised: *inflammatory pericarditis*, which is developed under the influence of some physical cause, and *rheumatic pericarditis*, which is developed under the influence of rheumatic disease. It is difficult to trace the progress and the duration of pericarditis; sometimes it goes through all its phases, and causes death in twenty-four hours; at other times, it lasts from twelve to fifteen days. The following is the general fashion of its progress: after severe premonitory symptoms, and sometimes without these, the patient is seized with pain in the region of the heart, fever and difficulty of respiration. Some hours after the attack, sometimes the next day, there is perceived a slight degree of friction sound, with or without vibratory tremor; the effusion becomes abundant in the first two or three days of the disease; towards the fifth or sixth day the febrile state diminishes, and the effusion becomes stationary; about the seventh or eighth day, absorption commences, and the friction sound is again perceived. In other respects, the progress and duration of this disease are subordinate to the intensity and extent of the phlegmasia, to its complications, to the causes which have produced it, to the age, temperament, &c. As relates to chronic pericarditis, it may, when once established, last for weeks, and even whole months.

Terminations. — Pericarditis may terminate in resolution, in adhesions of the pericardium, in the chronic state, and finally, in death. The termination by resolution is the most frequent, it is characterized by the absorption of lymph, and of the effused liquid. In some cases, there remains a little plastic lymph on the surface of the heart, which constitutes the *white plates* of the pericardium: these morbid products, when they are not very extensive, do not appear to be incompatible with a state of perfect health. We know that the disease inclines to resolution when the friction sound ceases, without leaving behind it more or less dulness, or a more marked impulse; all the other symptoms besides indicating resolution. But in a grave disease like pericarditis, it is not sufficient to be aware that resolution has commenced; we must also know all the signs which announce an amelioration in the state of the patient. These are only a diminution in all the general severe symptoms, and, moreover, a marked diminution in the dulness on

percussion. Should the pain, the fever, the anxiety, the strong impulse, and the pericardiac friction sound still persist, the inflammation progresses; but if the pain, from being fixed and pungent, cease entirely or nearly so, if the anxiety decrease, if the friction sound is no longer recognised, if the impulse of the heart diminish, and if there be only a slight acceleration in the beatings of this organ, it may be presumed that the inflammation is declining, without, however, our being able positively to affirm it.

The termination by adhesion constitutes a less happy termination than that by resolution. In general, the adhesions of the pericardium impede the action of the heart to such an extent as most generally to cause hypertrophy of the organ, and at a more distant period, death: when partial, they may, if not very extensive, be elongated by the movements of the heart in the pericardium, embarrass slightly, or not at all, the movements of the organ, and disappear after a certain length of time. This termination is announced by the following signs: 1st, cessation of a distinct friction sound; 2d, no augmentation in the dulness on percussion; 3d, violent impulse, simple and sometimes double kind, when the febrile state has completely ceased (but this only when the adhesions are general or very close). Acute pericarditis passes sometimes into the chronic state, when its nature is unknown, or when it is treated in an imperfect manner, especially if it affects persons of a bad constitution. This passage into the chronic state is marked by a modification in the intensity of the general symptoms, at the same time, and by the persistence of most of the physical signs (see *Chronic Pericarditis*).

The diseases which complicate pericarditis are by no means without influence over its termination. Thus, the inflammations of the pulmonary organs, or of the heart itself, often contribute to this result: endocarditis (which so often accompanies pericarditis) often leaves behind it, even after the resolution of the pericarditis, alterations about the orifices of the heart, which sooner or later cause death.

What shall we say respecting the terminations of chronic pericarditis? Here nature has to triumph, not only over the persistent inflammation, but, also, over the numerous morbid products accumulated about the pericardium. The termination by resolution is the most uncommon; that by adhesion would be desirable; but that by death is the most common, in a shorter or longer time, but generally not exceeding four months.

Prognosis. — There are, in our prognosis, two circumstances to consider — the intensity of the dyspnœa and the modifications of the pulse. If the dyspnœa be of an intensity disproportionate to the local symptoms, if the arterial beats be not in unison with the impulse of the heart, the disease is dangerous: if, on the contrary, there exist little or no dyspnœa; if the pulse preserve the normal characters and correspond to the impulse of the heart, the prognosis is favourable. The opinion of Corvisart on pericarditis is no longer

tenable; he regarded this disease as necessarily fatal, and only made an exception in favour of one form, which he termed *subacute*. It is now well known, that when acute pericarditis is timely treated, the prognosis is generally favourable, at least as regards life; most of the diseases of this nature yield to a well regulated treatment. The most difficult, as M. Bouillaud has remarked, are those which are complicated with endocarditis, carditis, a violent pleurisy, or pneumonia.

When pericarditis has terminated by adhesion, the final prognosis is unfavourable; but it is still more so, when pericarditis with effusion passes into the chronic state.

The prognosis is influenced by the age and temperament of the individuals who are affected with pericarditis: it is much more severe in those debilitated from former diseases; it often terminates the life of the patients who have for a long time been affected with a disease of the heart (hypertrophy, dilatation, and disease of the valves).

Treatment. — Acute pericarditis requires a very prompt and energetic treatment: all hesitation is dangerous, and the loss of a few hours is sometimes irreparable. If the disease is recent and acute, and if the strength of the patient permit it, a large bloodletting will be demanded, and as soon as reaction appears, twenty-five to forty leeches are to be applied over the præcordial region, one or two hours after the venesection. If the symptoms do not amend rapidly, the application of the leeches, and in some cases general bloodletting, two, three, or four times, according to the strength of the patient, at intervals of from eight to ten hours, or even as long as the state of the pulse and beats of the heart make us fear a reaction, are to be repeated. It will be necessary to join to this treatment, rest, diet, warm and demulcent drinks, and emollient applications over the painful part. It is not necessary in all cases to resort to so rigorous a treatment: often, a single application of leeches or cups over the præcordial region is sufficient to remove all the violent symptoms. Very often, aged patients, of a feeble constitution, or in whom the disease is very far advanced, cannot support a general bloodletting; in these cases local detraction of blood ought to be preferred, and thirty to forty leeches, or eight to ten cups, should be applied over the præcordial region. Finally, in some cases, especially in those of rheumatic pericarditis, in which the reaction is very feeble and the physical phenomena moderate, we may limit ourselves to the treatment which is directed for the primitive affection. We have seen pericarditis with rheumatism cured by the employment of nitrate of potassa in large doses, according to the method of Brocklesby and of M. Gendrin.

When, notwithstanding so energetic a treatment, the symptoms still maintain their intensity, we must have recourse to revulsives: blisters over the præcordial region, frictions with tartar emetic ointment and croton oil, and purgatives, may then be employed with advantage; mercurial frictions may be often had recourse to, in ac-

cordance with the practice of Hamilton. We have seen this latter treatment succeed so well in certain inflammations of serous membranes (peritonitis, for example), that we think it should not be neglected; but it must not be employed *parcâ manu*. It will be more successful when slight sensibility of the gums and soreness of the mouth, commonly called salivation, are produced.

During convalescence, the patient should be confined to a light and slightly stimulating diet, and should keep perfectly quiet until the movements and sounds of the heart have acquired all their normal characters. As relapses are not rare in pericarditis, it should be known that these secondary accidents have not the same intensity as the primitive ones, and that the danger of doing too much is greater than that of doing too little; a few leeches and blisters will compose the treatment.

In chronic pericarditis, we must have recourse to powerful revulsives, to repeated blisters, to cauteries (setons produce too much irritation). As the patient is always much enfeebled, he should have as much nourishment as possible; and if dropsy supervene, diuretics should be employed.

When, after having tried all the customary measures, the effusion does not yield, should we evacuate the liquid by means of a surgical operation? This has never been tried, and every prudent practitioner will shrink from its execution. Would not the pressure of the atmosphere on the organ be greater than that caused by the accumulation of liquid? And would this evacuation cure the chronic phlegmasia? We, therefore, reject this operation.

APPENDIX TO PERICARDITIS.

*Adhesions of the Pericardium.**

We have already seen that acute or chronic pericarditis often ends by the formation of adhesions between the folds of the pericardium. This termination, which is certainly less favourable than that by resolution, has, however, been regarded by many pathologists (among others by M. Bouillaud) as one which in no degree disturbs the play of the respiratory and circulatory organs, nor prevent the patients from enjoying the best health. Can it be that the heart should contract general adhesions, and consequently be considerably impeded in its movements, without a disturbance of the general economy? Such is not the opinion of Lancisi,

* We place here adhesions of the pericardium among the inflammatory affections, in order to keep up the continuity of the subject: they should, properly, be placed among the organic affections.

Vieussens, Senac, Meckel, Corvisart, Morgagni, and Hope, who have affirmed that health is incompatible with a complete and close adhesion of the folds of this membrane. Of what consequence is it that the patients appear to enjoy good health, and have fulness of habit, if on the least unusual exercise they are attacked with palpitations and dyspnœa? This is what is observed by patients themselves, when they give an accurate account of their condition. Most often, after an attack of pericarditis, they become incapable of working, and of efforts which they formerly accomplished with the greatest facility. Too frequently, certain patients, especially of the working classes, do not complain of these symptoms, because they have by degrees become habituated to a restricted circulation and respiration; but by questioning them with care, you will hear them say, that for some time they have had shortness of breath. As we have said, when treating of pericarditis, the termination by adhesion is not immediately fatal; but after a greater or less length of time, it becomes so, especially among the labouring classes, whose occupations are fatiguing, and whose life is irregular.

The adhesions of the pericardium are not only to be feared on account of the influence which they exert over the circulatory and respiratory functions, but, also, from the modifications which they induce in the nutrition of the heart. Whenever the adhesion is considerable, the heart is increased in volume, and acquires that form of hypertrophy which we shall describe under the head of *hypertrophy with dilatation*. For, independently of the part which the chronic inflammation may have in this complication, the heart is forced to redouble its energy, in order to strive successfully against the obstacle which the adhesions present to the exercise of its functions; consequently it becomes hypertrophied; and, besides, in consequence of the obstruction in its movements, there results a congestion greater than usual, and a distension of the cavities which, at first temporary, is not long in becoming permanent and producing dilatation.

When the adhesions of the pericardium have caused the formation of a hypertrophy with dilatation, they become confounded with this disease, whose gravity they increase; and, as it is impossible to separate them from the alteration which they have produced, we can at most only suspect their existence by the progress of the disease, and by the knowledge of the antecedent symptoms.

The diagnosis of simple adhesions of the pericardium has been always regarded as very obscure. Experience has not confirmed the exactness of the sign mentioned by Dr. Sanders, viz., that in this disease there is formed, in the epigastrium, below the left false ribs, a kind of depression resulting from the retraction movement of the diaphragm, at the moment of the ascension of the heart. As Laennec has remarked, in order that this depression should be produced, the stomach must adhere both to the diaphragm and to the abdominal parietes; a condition of things which does not exist.

Whatever may be the inherent difficulty of this diagnosis, we think, with Hope, that independently of the facts furnished by the antecedent symptoms of the patient, that is to say, the existence of pericarditis at a former period, and also the absence of a complete and extensive dulness of the præcordial region, as well as of the friction sounds belonging to the pericardium, and of this distance of the sounds of the heart, which form the principal bases of the diagnosis of pericarditis, we may advantageously make use of the following signs: 1st. A considerable arching of the præcordial region, with slight increase of dulness, without *displacement of the apex of the heart;* a phenomenon which is caused by the heart, whilst retained by its adhesions, not being able to descend; and besides, being arrested behind by the vertebral column, it naturally would force out the costal cartilages by its augmentation. 2d. The existence of a sudden movement, of a simple or double shock, which appears owing to the sudden arrest of the advancing and returning movements of the heart either by the vertebral column, or by adhesions.

CHAPTER II.

CARDITIS, OR INFLAMMATION OF THE MUSCULAR SUBSTANCE OF THE HEART.

Inflammation of the muscular texture of the heart is general or partial.

1st. *General carditis*, with general infiltration of pus, is excessively rare. Thus, Laennec, who regarded the production of pus as the only infallible sign of carditis, and who had never seen it, thought that there had not perhaps existed a single incontestable and well described case: but the beautiful observations related by Dr. Latham, and more recently those of M. Simonnet, leave no doubt on this point. A brownish colour of the tissue of the heart with diminution of its normal consistence, disseminated ecchymoses, infiltration of the muscular fibres with true pus, are the signs which belong to general carditis. Most pathologists are far from holding the circumscribed ideas of Laennec on the subject of carditis; for, according to them, the softening and induration, whatever be their colours, are most often incident to this disease. Do we not see inflammation induce in other muscles alterations of the same nature? Do we not see in pericarditis the heart present, in some isolated points on its external surface, alterations of this kind? We, therefore, think that general carditis is not so rare as Laennec supposed it to be, and we are very much disposed to refer to this disease the oftening and induration. However, as some authors

entertain doubts of the identity of the nature of these alterations, we have thought proper to reserve a particular chapter to each of them, in the series of the organic affections of the heart.

It is very difficult to assign to general carditis proper and exclusive symptoms, since this affection never exists without some lesions either of the endocardium or pericardium.

The symptoms ordinarily referred to it, are the following: the disease begins by a peculiar uneasiness of the præcordium, with slight pain in this region; palpitations, at first fugitive, and of slight intensity, soon followed by irregular movements; but notwithstanding these symptoms the sounds of the heart are not notably altered, except in their intensity; the dulness of the præcordial region being in its normal condition. Fever is soon lighted up; the pulse becomes strong, full, and resisting; there is difficulty of respiration, with orthopnœa, and extreme anxiety. To this reaction succeeds a period of depression; the sounds of the heart, before so violent, become intermittent; the difficulty of circulation is extreme; the face is flushed; the extremities are œdematous and cold; the dulness of the præcordial region increases in consequence of the congestion of the heart; the lungs are gorged; the pulse is feeble and fluttering; and the patient dies by a species of asphyxia.

Respecting the prognosis and treatment of general carditis, the remarks which we have made on pericarditis, and which we shall make on endocarditis, its two general complications, will suffice.

2d. *Partial carditis* is characterised sometimes by softening or induration of the muscular tissue of the heart, with or without discoloration, and only occupies some points of the heart; sometimes by the existence of *abscesses* or *ulcerations* on the internal and external surfaces of the heart. *Abscesses* are much more rare than ulcerations, although some authors mention a few cases. These purulent collections most generally occupy the left ventricle; their size has no limit, as they are sometimes of the size of a small walnut, at other times of that of a pea: they ordinarily consist either of a true phlegmonous or a caseous pus, of the consistence of a boiled white of an egg, or pus slightly reddish and of a fetid odour. The pus is sometimes found in contact with the fleshy tissue of the heart; sometimes it is surrounded by a true cyst. These abscesses may open into the ventricular cavity, or into that of the pericardium, and even into both at once. In the first case, the blood is mixed with the pus contained in the cavity of the abscess, and often produces a true aneurism of the heart; in the second case, the pus is poured into the pericardium, and death is the consequence. As regards the *ulcerations*, which depend on inflammation, they occupy much more rarely the external than the internal surface of the heart, and they often invade the muscular substance. They are met with much more frequently in the left than in the right cavities; they principally occupy the ventricles, and especially the valves; they may be single, or so numerous that they almost touch by their edges. Their extent varies also; they are ordinarily from

one to two lines in diameter, but they have been seen to invade the whole left ventricle, and destroy the septum. Their depth varies no less; they may be superficial, and may stop by laying bare the muscular substance, which in this case is covered by a light bed of pus, or studded with fungosities; or they may be from a quarter to half a line in depth; in this last case their bottom may be of a scarlet red colour, like the spot in purpura, or greyish, or black, lined by a false membrane, or stained by blood. The ulcerations which occupy the external or internal surface of the ventricles may induce, in the last stages of disease, a rupture of the heart; while those which occupy the internal surface may give rise to a true aneurism of the organ. Ulcerations of the valves of the heart may perforate them, and produce regurgitation. Finally, in some rare cases, the ulcerations which occupy the septum of the ventricles may form a communication between the right and left cavities, and consequently produce *cyanosis*.

Abscesses and ulcerations of the heart possess no sign by which they can be distinguished from other affections of this organ.

Ruptures of the heart are owing most frequently to ulcerations. When they result from other causes, it is ordinarily owing to a disease which has diminished the cohesion and thickness of the muscular tissue, such as softening, fatty degeneration, &c.: they are commonly in the left ventricle, because it is strong, and contracts with more energy; for muscles often break by the force of their own contractions. These ruptures rarely take place about the apex, contrary to what would be supposed, considering the slight degree of thickness of this part of the organ. The direction of the rent varies: it is transverse, or oblique, or rather vertical, and parallel to the muscular fibres. Its extent varies also; it may be from a few lines to two or three inches in length. The rupture is always arrested by the fibrous tissue of the heart; the tendons and orifices always escape. The lips of the wound may be clean and regular, or very irregular, jagged, and anfractuous; its internal orifice is always more narrow than the external; in some cases, it is closed by a sanguineous concretion. The ruptures of the heart may be simple or numerous; as many as five have been met with in the same individual. We find at the same time, in the pericardium, an abundant effusion of blood, partly coagulated, which lines the heart, and conceals the solution of continuity.

These ruptures are most frequently produced by causes depending on violent efforts, paroxysms of anger, external violence, &c. They are announced by the following signs: pungent pain in the præcordial region; loud cries; great paleness; instinctive contraction of the extremities; syncope, though sometimes this affection comes on later; and the invalid, after having remained more or less time in this state, is seized with symptoms of reaction, which cause death in a few hours. According to many authors, in more fortunate cases, all the symptoms are observed to lose their severity; the respiration and circulation are established; the blood is absorbed;

a cicatrix forms at the point of rupture, and a cure takes place in fifteen or twenty days. As ruptures of the heart kill almost instantaneously, it is always difficult to recognise the disease before the patient has died; however, when death does not take place so rapidly, it may be suspected from the presence of the preceding signs, and of those which announce an effusion into the pericardium. Ruptures can only be confounded with sudden apoplexy, ruptures of the diaphragm, rupture of the large vessels of the chest, or with syncope. Apoplexy, whatever be its intensity, does not immediately stop the beatings of the heart, and these preserve their normal character; ruptures of the diaphragm do not kill in so sudden a manner; syncopes do not prevent the pulsations of the heart from being heard, and they cease often, in a certain space of time, by posture and stimulants, &c.; there remains a rupture of the large vessels, especially in the pericardium, which it is impossible to distinguish from rupture of the heart, otherwise than by the antecedent symptoms.

Rupture of the heart may interest not only the muscular parietes, but also the *columnæ carneæ* and *chordæ tendineæ* of this organ. It is by violent efforts, also, that it is caused. The symptoms which announce it supervene suddenly; at the same moment, the patients are attacked by extreme dyspnœa, by paleness, by coldness of the extremities, and soon by all the general phenomena belonging to diseases of the heart. This group of symptoms is so remarkable, that they may be considered as indicative of rupture, especially when they appear after violent exertion. It is in general very difficult to recognise the seat of rupture, unless we are able, by means of the physical signs belonging to disease of the valves, to form our diagnosis.

Shall we speak of gangrene of the heart? We have never observed it as a termination of inflammation of the tissue of this organ, and it appears to us impossible that it can take place; first, because the muscular tissue is not very susceptible of mortification, and, also, because the inflammation would prove fatal before producing gangrene.

CHAPTER III.

ON ENDOCARDITIS.

The name of endocarditis has been given to inflammation of the internal membrane of the heart. This affection, for a long time unknown, had been noticed by Corvisart, Frank, Kreysig, Laennec, &c.; but it is principally M. Bouillaud who first fixed attention on inflammation of the internal membrane of the heart

and great vessels, and demonstrated the coincidence between this disease and acute articular rheumatism.

Endocarditis, like all phlegmasiæ of membranes, may be acute or chronic. We shall only describe here acute endocarditis; what regards the chronic variety, will be found in the chapter on *Diseases of the Valves.*

Anatomical Characters. — As acute inflammation of the endocardium very often coincides with acute arteritis, it will be proper to describe simultaneously the anatomical alterations in these two diseases. We refer to the following chapter for the anatomical characters of chronic arteritis.

1st. *Alterations of the Serous Membrane and Subjacent Cellular Tissue.* — Redness of the internal membrane of the heart and arteries is most commonly represented to be an index of an inflammation of these vessels; it may, however, be wanting when the inflammation has not been long enough to cause the stasis of blood. This redness is most often partial, and in this case it principally occupies the valves. It presents a more or less bright tint, sometimes of a rose colour, sometimes scarlet, sometimes of a violet or brownish colour; most generally it is not uniform, and it decreases gradually about the borders: it is generally accompanied by injection of the subjacent capillary vessels of the internal membrane, which is especially well-marked in the arteries. The nature of this redness has given rise to numerous discussions: it has been a matter of question whether this inflammatory redness can be anatomically distinguished from that produced by sanguineous imbibition. The numerous experiments of Laennec, and of his successors, have definitively fixed the value of this phenomenon; and it is now perfectly well established, that this redness is not sufficient, *of itself*, to characterize the inflammatory nature of the disease, unless some other characters appertaining to inflammation are also found; and that it loses all its importance when it is found after a long struggle, in the case of manifest alteration of the blood, and in that of advanced putrid decomposition. Thickening of the internal membrane is especially observed at the valves; most commonly it is only apparent, and is due to false membrane situated above or below the internal membrane; which latter loses its transparency and becomes softened. Its external surface rests on the softened cellular tissue, which is sometimes infiltrated with plastic matter; and hence this membrane can be detached with great facility. Finally, ulcerations may be found on the internal face of the heart and large vessels, without any lesion of the subjacent tissues.

2d. *Products of Secretion.* — As the heart and arteries are the seat of a continual circulation, all the products of inflammation ought to be, in a great measure, carried away by the force of the column of blood. There is, however, sometimes found an effusion of plastic matter on the free or adherent border of the valves of the aorta. Baillie, Laennec, and Hope say, that they have found it

even on the free surface of the internal membrane of the auricles and in the ventricles, also. The plastic matter is at first slightly adherent to the internal membrane, but it is readily organised and united intimately with the vessel. After all, these false membranes are very rare. As to the pus, whose liquidity disposes it still more to be borne away by the column of blood, it is often found in the centre of a clot, or is retained in the meshes of the *columnæ carneæ* (M. Bouillaud); or, again, in the cellular tissue subjacent to the internal membrane: it is very rare to find pus in the arteries.

3d. *State of the Cardiac and Arterial Tissue.*—It is unusual to find the muscular substance of the heart sound in endocarditis; presenting, as it always does, some modification in its colour and consistence; sometimes it is infiltrated with pus or blood. Pericarditis very often coincides with endocarditis, and presents its ordinary anatomical characters. The arterial coats often participate in the inflammation of the internal membrane, and we find, independently of the injection of the *vasa vasorum*, considerable fragility of the fibrous and cellular membrane of these vessels.

4th. *Polypous Concretions.* — Coagulation of blood takes place in the interior of the heart in endocarditis, in the same manner as it does in the veins when affected with phlebitis. The concretions exhibit in the heart as well as in the arteries the form of white, discoloured, elastic, glutinous clots, adherent to the arterial or cardiac parietes, and twisted around the valvular cords and *columnæ carneæ;* they are about half organised, and very analogous to the inflammatory clot of the blood, or to false membranes themselves. Some have points or red lines, which are only the rudiments of vessels. These concretions differ much as regards their size and configuration; when they occupy the heart, they generally extend into the large vessels, and are almost always more voluminous in the right than in the left cavities.

Their closest adhesion is commonly about the free border of the valves, where some fragments will be found after repeated washings (Bouillaud).

It is useless to add, that, if the resolution of the disease be not complete, the thickening of the internal membrane and of the subjacent cellular tissue becomes permanent, the false membranes and clots which adhere to the surface become organised, and all these accidental productions may undergo analogous transformations. Whatever else there is relative to the anatomical characters of chronic endocarditis, will be found in the article on the *Anatomical Characters of the Diseases of the Valves.*

Causes. — The causes of endocarditis, like those of pericarditis, are *mechanical, physiological or pathological.* The first comprehend falls, blows, compression on the præcordial region, wounds of the heart, &c.; the second embrace all the causes which strongly excite the circulatory functions, as youth, exciting nourishment, plethora, &c.; we place, lastly, in the third set, sudden suppression of the perspiration, extension of inflammation of the lungs, of the

pleura, or large vessels; certain diseases which result in more or less alteration of the blood (purulent infection, typhoid affections, eruptive fevers, perhaps syphilis); the action of certain medicinal agents; finally, acute articular rheumatism (the prominent and powerful cause noticed by Bouillaud).

Symptoms. — A. *General Symptoms.* High *febrile reaction*, with dryness of skin and great thirst; the *posture* of the patient, without being as constrained as in pericarditis, is always attended with certain difficulty and anxiety, which amount to the most insupportable anguish when the circulation is embarrassed; *face* injected, though but little altered; it is neither purple nor swollen; *respiration* slightly accelerated; but when the patient is quiet, there is little or no oppression; pulse full, strong, hard and regular: its frequency varies from 90 to 120 pulsations. Such are the general signs which announce endocarditis at the beginning, and whilst the circulation continues to move freely. There is, however, a certain number of cases in which we meet with some symptoms which will soon be enumerated; such as feebleness, irregularity, intermissions, inequalities in the pulsations of the heart, orthopnœa, anxiety, although no mechanical cause can be found to account for them. But these symptoms only exist for a short time, and are never well marked. We have already seen that something analogous is observed in pericarditis; and it is probable that these symptoms are developed, in both circumstances, under the influence of a nervous disorder, or an increase of irritability.

When the cardiac circulation is much impeded, whatever may be the cause (alterations of the valves, or polypi of the heart), we see that series of grave symptoms which always indicate considerable embarrassment in the circulation: the *pulse* becomes feeble, irregular, unequal, intermittent, and excessively frequent, from 140 to 160; it contrasts often by its characters with the extraordinary violence of the beats of the heart. (Sometimes there are two beats of the heart for one diastole of the pulse, because the heart projects too small a quantity of blood, for the undulation to be transmitted to the radial artery.) From this obstructed circulation there results a frightful *paleness*, extreme *anxiety*, with tendency to syncope, and suffocation, which force the patient to keep the upright posture; a *violet* or *livid* tint of the integuments; *œdema* of the face and extremities, with general coldness; some *disorder of mind*, and, later, continual *faintings;* slight convulsive movements, stertorous respiration, with frothing at the mouth, and, finally, death.

B. *Local Symptoms.* — 1st. *Physiological Symptoms.* The *pain* is not constant; but there is always an undefinable feeling of wretchedness, uneasiness or anxiety about the præcordial region. During the first stage, the pulsations of the heart are strong, full, and regular; but when the circulatory disorder supervenes, they lose all these characters, and become irregular, unequal, and intermittent: they sometimes do not accord, as regards force and number, with the pulsations of the arteries. Finally, the arching of the præcordial

region has been noticed as a sign; but it is impossible to admit its existence, unless there be at the same time an effusion into the pericardium.

2d. *Physical Symptoms.* — *Percussion.* The dulness on percussion is increased; according to Bouillaud, it may have an extent of 4, 9, and even 16 square inches. It is difficult to explain this increase of the volume of the heart, unless we attribute it, as Hope does, to the distension of the cavities of this organ by blood or polypous concretions. We, therefore, are induced to believe, in accordance with this last author, that, in endocarditis, the dulness only increases slightly unless the cardiac circulation be considerably embarrassed. This dulness, when it exists, is distinguished from that which is owing to an effusion into the pericardium, by the impulse of the heart, in the former, raising *sensibly* the parietes of the chest, and being synchronous with the first sound; whilst, in the case of effusion, the impulse is indistinct, undulatory, and not synchronous with the pulse. In the former, the sounds of the heart are perfectly clear and distinct, and not distant; whilst in effusion, they are feeble, stifled, and, as it were, distant; finally, in the former, the dulness extends principally in a transverse direction, whilst in pericarditis, it is principally upwards.

Auscultation. — Auscultation shows a *murmur* (blowing sound), which accompanies a single sound of the heart, or both at once. If this murmur coincide with the first sound, we conclude that the inflammation has given rise to a contraction of one or the other arterial orifice, or to a deficiency of the right or left auriculo-ventricular valves, perhaps to both affections at the same time: if it coincide with the second sound, we may conclude that there is a deficiency of the semilunar or aortic valves, and perhaps a contraction of one or other of the auriculo-ventricular orifices. It is a rare event for the contraction of these last orifices to be of sufficient extent, in acute endocarditis, to give rise to a murmur. Moreover, in the immense majority of cases, the alterations and murmurs are limited to the left ventricle. (See, for further details, the chapter on the *Modifications of the Sounds of the Heart* — article *Intra-cardiac Murmurs;* and the chapter on *Diseases of the Valves* — article *Physical Symptoms.*)

When polypous concretions almost entirely obstruct the heart, we scarcely hear the anomalous sounds, and the sounds of the heart themselves lose their sonorous character. (See *Polypi of the Heart.*)

Diagnosis. — The numerous details we have given already on the differential diagnosis of pericarditis, and the multiplied points of resemblance that exist between this disease and endocarditis, leave us little to say in this place. As a general rule, when called to a patient presenting, 1st, a violent febrile reaction; 2d, an active impulse of the heart; and, 3d, an intra-cardiac murmur (blowing sound) which did not exist before, we may admit the existence of endocarditis.

There is one point on which we insist; — it is the differential

diagnosis of pericarditis and endocarditis; independently of some differences in the general symptoms (as the greater intensity of pain in pericarditis, and the rapidity with which dangerous complications supervene), the differential diagnosis of these two diseases rests, in a great measure, on the dull and stifled character which the sounds of the heart present in pericarditis, and on the differences which exist between the friction sounds belonging to the pericardium, and the valvular murmurs. Now is the time to make known the differential characters of these two sounds;

Valvular Intra-cardiac Murmurs.	*Friction Sounds.*
1st. The valvular murmurs are always deeply seated and distant.	1. The friction sounds are always superficial, and take place immediately beneath the ear.
2. They are commonly simple.	2. They are most often double.
3. They generally have a soft character (that of the bellows sound), at least in acute endocarditis.	3. They are rough, and when they coincide with the valvular murmurs, they are perceived, in some measure, through the medium of these last.
4. The murmurs with the second sound, or diastolic, are very seldom rough.	4. The diastolic sounds which present rough characters are probably friction sounds.
5. The valvular murmurs once established, very slowly change their nature, and never their seat.	5. The friction sounds rapidly and frequently change their nature and their seat.

Forms, progress, duration, and *terminations.* — Endocarditis may present *three* well-marked *forms;* the *inflammatory* form, which is most often developed under the influence of physical causes, and also of suppression of perspiration; the *rheumatic* form; and, finally, the *putrid* or *typhoid* form, which takes place in those diseases where the sanguineous fluid is altered. Its *progress* may be simple and regular; it may go through all its stages in the ordinary space of time, or may be delayed by accidents or complications.

When endocarditis is subjected early to an active and well directed treatment, it is not unusual to see it terminate happily in four or five days: its ordinary duration is from six to eight. It may terminate by complete resolution, that is, without any trace of the valvular murmurs; but when these murmurs are prolonged beyond eight or ten days, there is danger of their continuing for many weeks, and even becoming permanent, and of the endocarditis passing into the chronic state. This termination by disease of the valves is the most common, because the patient is not submitted to a treatment sufficiently prolonged, and because the severe disease (acute rheumatism), which is almost always the cause of acute endocarditis, is treated too lightly. Termination by death is not common.

Prognosis —The prognosis varies according to a variety of circumstances, dependent either on the individual affected, or on the disease itself. It is in general very favourable, at least in relation to the immediate danger to the life of the patient. It may, how-

ever, terminate by death in two or three days; but endocarditis is especially dangerous, by its leaving often behind it organic alterations, which increase in a gradual though scarcely appreciable manner, embitter the life of the patient, and necessarily cause death.

Treatment. — The treatment of acute endocarditis ought to be neither less prompt nor less rigorous than that of pericarditis. We must not be misled by the apparent lightness of the symptoms, and we should never forget that an organic disease of the valves, whose development it is necessary to guard against, may supervene.

When called to a case of endocarditis in the commencement, if the patient is well constituted and young, the antiphlogistic treatment should be rigorously made use of; repeated bleedings, after the method of M. Bouillaud, will be required, and produce the best results. When the patient is feeble, or very old, when he is affected with a former disease, or under the influence of some debilitating operation, it is proper to try only one general bleeding, and to renew local bleedings (leeches, cups); in some cases where endocarditis is only a very feeble element of the disease (as in certain acute articular rheumatisms), we may, when the general symptoms are not severe, limit ourselves to the treatment of the primitive affection. We have seen rheumatic endocarditis cured under the treatment directed against the rheumatism.

When we have exhausted, without any beneficial result, all the antiphlogistic measures against endocarditis, such as general and local bleedings, diluting drinks, cataplasms, general baths, rest, and diet; or when fulness, or some particular conditions contraindicate their application, it is proper to have recourse to repeated blisters over the præcordial region. This is an excellent measure, and if we place on the denuded surface of the skin from 30 to 40 centigrammes of powdered digitalis, we often notably diminish the feeling of uneasiness which the patient experiences in the region of the heart. It is almost always proper to add to the employment of blisters that of revulsives to the intestinal canal; and finally, when there is danger of the endocarditis passing into the chronic state, a deep cautery, which will suppurate and keep open, over the region of the heart, in order to exercise a powerful revulsion, at the same time that the patient is placed in a state of absolute rest, and submitted to cooling regimen. It is in this last case that we can have recourse to the treatment of Hamilton; that is, mercurial frictions over the region of the heart, and, internally, mercury combined with opium; digitalis, and hyosciamus should also be used internally.

CHAPTER IV.

ON ARTERITIS.

Arteritis is an inflammation of the arterial tubes: it may be acute or chronic.

Acute Arteritis.—We have not much to say respecting acute arteritis, and especially respecting the acute inflammation of the large vessels arising from the heart. We have described, in the article *Endocarditis*, the anatomical alterations which are common with those belonging to this last disease. It is extremely rare to meet with acute inflammation of the large vessels, independently of that of the internal membrane of the heart. It is always very difficult to be recognised, because these vessels, by their position, are almost entirely beyond our investigation. We may, however, suspect inflammation of the aorta when we find its beats increased in force, pains, which are dull, and deeply seated, like those of rheumatism, along the course of this vessel, with accompanying sensation of heat and uneasiness. If to these symptoms be added the presence, in this artery, of a murmur more or less rough, short and sudden, which was not heard before, and the group of symptoms which have been described under the name of *inflammatory fever*, we may admit, with some degree of certainty, the existence of *aortitis*. The inflammation may affect the arch of the aorta separately, the pectoral or abdominal aorta; it may commence in endocarditis, and thence extend itself to the rest of the aorta; or it may commence at a more or less distance from the heart (in an extremity, for instance, after the ligature of an artery), and thence extend towards the heart. As to *inflammation of the pulmonary arteries*, it is much more difficult to recognise than acute aortitis; Broussais noticed, as characteristics of this inflammation, the signs of hypertrophy of the right cavities of the heart, with a feeling of burning heat in the chest, or the existence of an inflammation, or obstinate pulmonary hemorrhage, with violent beatings and very acute sensibility. There is no necessity for our insisting on the slight foundation for these signs. We believe, that it is sufficient, in order to make sure of the existence of this inflammation, to find, together with a collection of grave general symptoms, a more or less rough murmur along the well known course of this artery, having the characters which ordinarily belong to arterial murmurs. To speak further of the causes, prognosis, and treatment of these diseases, would be only to repeat what we have said of endocarditis itself.

Chronic Arteritis.—Chronic is much more frequent in the great vessels than acute inflammation. The anatomical alterations belonging to the disease may affect the internal membrane, the sub-

serous cellular tissue, the intervening membrane, sometimes the cellular membrane of the artery, and most generally all these tissues at once.

Alterations of the membranes consist principally in a deep red or dirty-red colour of the internal membrane, and considerable thickness with diminution in its consistence, loss of transparency, and anomalous facility of detachment from the subjacent tissues. All the arterial coats generally participate in the thickening; they become hypertrophied by losing their elasticity, and are gradually dilated. The presence of newly formed products imparts a new form to the arteries; the subjacent cellular tissue becomes infiltrated with an opaque, straw coloured matter, which resembles in its consistency yellow wax or cheese (*steatomatous* productions), or a white matter, having the consistence of a friable paste, sometimes like pus; at other times a substance having a fibrous or ligamentous appearance (*fibrous productions*,) or transparent, white, and elastic (*fibro-cartilaginous* or *cartilaginous* productions). Soon, in the middle of these cartilaginous or fibro-cartilaginous flakes, at other times in the midst of steatomatous productions, calcareous products are deposited, of an irregular form, as mastic or mortar, or resembling laminæ and scales. These calcareous productions differ essentially from the natural osseous tissue; and even when they are developed in the middle of cartilage, they never present either organisation or the particular arrangement of the osseous tissue; it is a kind of irregular and homogeneous crystallisation without any determinate arrangement, without proper vitality, and which only contains a very small quantity of animal matter (phosphate of lime, 65·5; animal matter, 34·5). These morbid productions, especially those which are calcareous, are almost always followed by the destruction and ulceration of the internal membrane. Ossifications* may occupy a part or even the whole circumference of the artery, may limit themselves to a

* Can it be true that, in every case, the morbid productions whose history we have just traced, are the result of a chronic inflammation? May it not be admitted, that some of them, and, in particular, calcareous productions, can be produced independently of inflammation? Is not their so frequent appearance in old persons, their often irregular dissemination, the absence of any alteration of the membrane which covers them, sufficient to establish the non-inflammatory nature of these alterations? The cause, in this case, would remain to be known. Might it not be in the depravation of action of the arterial tissue taking place, as Hope thinks, under the influence of the excessive distension which the arteries experience from the force of the circulation? It is known that, when pressure and habitual friction are made on certain parts, an induration at length results: for example, the horny excrescences of the feet caused by too narrow shoes; callosities of the hand produced by hard labour. What is true of other parts, may be also true of the arterial tissue. Indurations are most often present in the arteries that are the most exposed to an extreme distension, as the arch of the aorta, which sustains by itself the whole impulse of the ventricle, the arteries of the brain, which are deprived of cellular membrane. They are also especially met with in old persons whose arterial, like every other tissue, has lost its elasticity and cohesion. We do not express a decided opinion; but an important fact remains to be ascertained; degenerations of the arteries, and especially ossifications, do not always acknowledge an inflammatory cause.

small extent of the arterial system, or transform a part or the whole of this system into an osseous, inflexible tube; frequent in the aorta, they are very rare in the pulmonary artery. The ulcerations which most often result from calcareous incrustation, of which we have just spoken, vary in extent from a line to an inch; generally round, they may be irregular, and sometimes only consist of a transverse slit, closed often by a discoloured clot; their edges are more or less thick and unequal; they present a dirty and sanious appearance; their depth varies; in some cases they are only confined by the external or cellular membrane. It is most generally, in consequence of these ulcerations, that the production of aneurisms takes place. In certain cases, the cartilaginous or osseous flakes, deposits of calcareous matter, &c., occurring in the interior of the artery, a considerable contraction of its caliber results, and this contraction may even be so great as to cause complete obliteration. In relation with this state of coarctation of the arteries, are the cases in which the arterial tubes, deprived of elasticity, are dilated by the impulse of the blood, constituting alterations, of which we shall speak further on, under the head of *Aneurism by Dilatation.*

The *causes* of chronic arteritis are not well known; it often succeeds to acute arteritis, in which the treatment has not been properly directed, or which was developed in persons of a weak or bad constitution. Arteritis may be chronic from the beginning, and be owing to chronic causes. Among these we would place violent and repeated muscular efforts, certain professions, and especially hypertrophy of the left side of the heart, whose continual action is mainly spent on the arterial tubes.

The *symptoms* of chronic arteritis are very obscure when it has not produced any great alterations in the texture of these vessels. It is probable that this disease deranges the general health; but as it always causes a pathological state of the heart, it is difficult to tell its separate share in the production of morbid alterations. When the arteries are placed within the reach of our means of investigation, we may recognise their chronic inflammation by the modifications which they undergo in their pulsations and sounds (very feeble dilatation, induration, vibratory tremor, more or less rough murmur). When, on the contrary, the vessels are placed beyond our inspection, as the aorta and pulmonary artery, we can no longer make use of auscultation. However, if we perceive, in such cases, murmurs along the course of these vessels, we may suspect the existence of chronic arteritis; especially if the patient had formerly a cardiac or arterial phlegmasia, and if the symptoms are entirely circumscribed to one point of the arterial system.

Chronic arteritis is a very dangerous disease; for the alterations which it produces have, as inevitable results, first dilatation, and afterwards rupture, or obliteration of the arterial tube. We refer, for the treatment, to *Diseases of the Valves*, and to *Aneurisms of the Aorta.*

SECOND CLASS.

ORGANIC DISEASES OF THE HEART AND GREAT VESSELS.

CHAPTER I.

HYPERTROPHY OF THE HEART.

The name of hypertrophy is given to an increased thickness of the muscular texture of the heart, resulting from an increase in the nutrition of this organ. This affection, which Corvisart had already investigated under the name of *active aneurism*, has only been well understood since the labours of Bertin in 1811.

Hypertrophy presents the following varieties:

1st. *Simple hypertrophy*, in which the parietes are thickened and the cavity dilated.

2d. *Hypertrophy with dilatation* (*eccentric or aneurismal hypertrophy* of Bertin), which comprehends two varieties:

First variety, with dilatation of cavities, and increased thickness of the walls.

Second variety, in which the parietes present their natural thickness, and their cavity is dilated (*hypertrophy from increased extent of the walls*).

Hypertrophy with contraction (*concentric hypertrophy of Bertin*),* in which the walls are thickened and the cavity diminished in extent.

Before describing the anatomical characters of hypertrophy of the heart, we should give an idea of the natural dimensions of this organ: "The heart, including the auricles, should have a size which is inferior, equal or slightly superior to that of the fist of the subject; the walls of the left ventricle should have a thickness more than double that of the parietes of the right ventricle. Their tissue being more compact and firm than that of the muscles, ought to prevent them from collapsing, when the ventricle is opened. The right ventricle, a little larger than the left, with the *columnæ*

* With Doctor Hope we have preferred the names *hypertrophy with dilatation and contraction* to those adopted by Bertin, *eccentric and concentric hypertrophy*, which are much less simple and less exact; besides, this last nomenclature has the inconvenience of designating by two different names (*true aneurism and eccentric hypertrophy*), two affections whose nature is entirely identical. As these two only differ in the relative degree of hypertrophy and dilatation, it is easy to avoid the difficulty,—by placing the words *dilatation* or *hypertrophy* first, according as one or other of these affections predominates. Thus, *hypertrophy with dilatation* indicates the predominance of hypertrophy, whilst *dilatation with hypertrophy* indicates predominance of dilatation. *Hypertrophy by increase in the extent of the walls* (without alteration in thickness), only takes this name when it coincides with the symptoms of hypertrophy: it takes the name of *simple dilatation* when the symptoms are those of dilatation.

carneæ more voluminous, notwithstanding the inferior thickness of its walls, ought to collapse after incision."

In the fœtus, and very young children, the left ventricle has not its walls proportionately as thick as in the adult. The greater capacity of the right ventricle is not owing to the sanguineous distension which takes place in this cavity during the act of dissolution; for this disposition is also found in animals who have died from hemorrhage.

For the inexact rules furnished by Laennec, M. Bouillaud has substituted truer calculations, which are drawn from the measurement and weight of the heart. Although they are not substantiated by a great number of facts, they still deserve to be taken into consideration. In an adult of ordinary size, and well formed (he says), the medium weight of the heart is from eight to nine ounces; the mean circumference of this organ, at its base, is from eight to nine inches; the mean longitudinal and transverse diameter is about three and a half inches (the transverse diameter generally gains a little on the longitudinal); the measurement of the antero-posterior diameter is about two inches; the mean thickness of the walls of the left ventricle at its base is from six to seven lines; the mean thickness of the walls of the right ventricle at its base is about two and a half lines; the mean thickness of the walls of the left auricle is one and a half lines; and the mean thickness of the walls of the right auricle is one line; the ventricular cavity, on an average, may contain a hen's egg; the cavity of the right ventricle slightly exceeds that of the left ventricle.*

Anatomical Characters. — Hypertrophy may affect only one cavity, or many cavities of the heart at the same time. Under certain circumstances the walls of one cavity are thickened, whilst those of the other are thinned. The ventricles are much more subject to hypertrophy than the auricles.

1st. *General Hypertrophy.* — When all the cavities of the heart are hypertrophied, especially when they are dilated at the same time, the heart is capable of acquiring a size which is three or four times more considerable than in the healthy state; and its weight, which is ordinarily from eight to nine ounces, may be three times

* MM. Bizot and Clendinning have criticised the results of M. Bouillaud relative to the measurement and weight of the heart. According to M. Clendinning, the heart continues to increase in weight to a very advanced age. These are his measurements:

Weight of the heart.	Medium.	Males.	Females.
From 15 to 30	. .	8¼	8¼
30 to 50	. .	8½	8½
50 to 70	. .	9⅓	8
70 years and upwards		9¾	8

The investigations of M. Bizot nearly agree with those of M. Clendinning; but M. Bizot, in some measure, renounces them when he asserts, "*that he had not considered as diseases of the heart those alterations which exist without giving rise to the recognised symptoms of the diseases of this organ.*"

as much. In proportion as this organ increases in size, it loses its oblong form, becomes spherical, and sometimes even broader than it is long: its apex is scarcely distinguishable; at the same time, in consequence of the resistance which it meets with, by the diaphragm preventing its descent, it takes a more horizontal position, encroaches upon the left cavity of the chest, and sometimes forces the border of the lung on a level with the inferior edge of the fourth rib. The heart being enlarged in its size, obeys the force of gravity, unless retained by the adhesions of the pericardium.

2d. *Hypertrophy of the Left Ventricle.* — This ventricle is much more susceptible of hypertrophy than the right one. When it is hypertrophied, it extends over the whole præcordial region; sometimes so far beneath the sternum as to confound the impulse of the left ventricle with those of the right: its walls, which are, on an average, about half an inch thick, may then attain the thickness of one and a half or two inches. The greatest degree in this respect is generally observed a little above the middle of the ventricles, on a level with the insertion of the *columnæ carneæ;* this thickness diminishes in proportion as we approach the aortic orifice or the apex. When the hypertrophy is simple, it only increases the regular form of the ventricle without altering it; but when the hypertrophy takes place within and diminishes the cavity, the heart assumes a globular form; sometimes the *columnæ carneæ* are hypertrophied; sometimes they are tense and flattened, especially when the dilatation is considerable. The inter-ventricular septum generally participates in the hypertrophy, but in a less degree than the external walls. The right ventricle, when it does not participate in the hypertrophy of the left ventricle, is attached to the superior and lateral part of this ventricle; it appears flattened and diminished in size; and when it is a little elongated (which is most often the case), it is, as it were, folded around the left ventricle. When the left ventricle is both hypertrophied and dilated, its cavity, which, on an average, can contain a hen's egg, is sometimes so much increased, that it will admit a large orange, or the fist of an adult. On the contrary, in hypertrophy with contraction, its cavity may be reduced to the size of a small walnut, or pigeon's egg.

3d. *Hypertrophy of the Right Ventricle.* — The right ventricle may descend below the left ventricle, and constitute of itself the apex of the heart: generally, the hypertrophy affects the walls of the ventricles themselves less than the *columnæ carneæ*, which are naturally more numerous and complicated than those of the left ventricle; in some cases it is limited to these last. The *columnæ carneæ* are sometimes interlaced and attached in so curious a manner, that they traverse the right ventricle in all directions, subdivide it into different compartments, and almost completely fill its cavity. This disposition is met with more rarely, and is less marked in the left ventricle. The thickness of the walls of the right ventricle, the medium of which is about 2½ lines, rarely exceeds 4 or

5 lines; it may, however, extend to 11, and even to 16 lines; it is ordinarily near the base of the right ventricle that the greatest thickness is found; below this, notwithstanding the hypertrophy of the *columnæ carneæ*, the walls are most frequently thin, and sometimes, between these columns, even transparent. When dilatation is joined to hypertrophy, the cavity of the right ventricle, which, in the healthy state, admits a hen's egg, will receive, in some cases, a goose's egg, and even more. When, on the contrary, contraction is added to hypertrophy, its cavity may be contracted so much as not to contain a pigeon's egg. It is rare to observe this last alteration, unless there are at the same time some malformations of the heart.

4th. *Partial Hypertrophy.* — This hypertrophy, which, as we have seen, is limited to one ventricle, may be still more partial, that is, limited to some points; for example, to the base, to the interventricular septum, to the apex, to the *columnæ carneæ*, or to the external walls, whilst the rest of the cavity remains sound. Moreover, a thickened ventricle may be contracted at one point and dilated in another. It is in these mixed cases, as Hope justly remarks, that we must counterpoise the opposite conditions; balance the hypertrophy against the attenuation, and the dilatation against the contraction, in order to determine which is the predominant affection.

5th. *Hypertrophy of the Auricles.* — This state almost constantly coincides with the dilatation of these cavities; although, simple hypertrophy and hypertrophy with contraction are not unexampled. It uniformly affects the whole extent of the walls: but it is ordinarily at its maximum in the appendices, and especially in the muscular bands; it also acquires a greater state of extension in the right auricle, where the *musculi pectinati* are larger and more numerous than in the left. This thickening may be so great, as to give to the right auricle the size of the right ventricle. As regards the left auricle, its thickness does not extend beyond its natural state, which is about a line and a half.

In hypertrophy, the muscular substance of the heart is generally more firm and red than in the natural state, but sometimes it is more flaccid and pale. Its increased firmness may be so great as to constitute *induration*, a distinct affection, which does not consist in mere augmentation, but in an alteration of nutrition, or an increase of density in the elementary molecules of the part.

When hypertrophy succeeds endocarditis or pericarditis, and likewise under some other circumstances, we find particular alterations of the membranes, such as adhesions, thickening or opacity of the valves and their *chordæ tendineæ*. In the case of considerable hypertrophy with dilatation, the valves are most generally thickened and opaque.

Causes. — Every time that a muscle takes on increased action, it receives an increased flow of blood; and consequently a proportional increase of nutrition results. What is seen in the arm of

blacksmiths, in the legs of dancers, is also seen in the heart. When this organ, from any cause, becomes over excited by extraordinary exertion, there is an increased activity in its proper circulation, and consequently increased nutrition. In proportion as the walls are thickened, its contractile power augments; and these effects being added to the exciting cause, increase still more the activity of the heart's action, and the disease in the same proportion. The left ventricle is more liable to hypertrophy than the right, and the right ventricle more than the auricles. The reason of this is plain; the resistance which the muscles oppose to the force that distends them, is exactly in proportion to their thickness. As we have said before, the act of resistance increasing the activity of the circulation, causes an increase of nutrition. Hypertrophy, therefore, should most often take place in those muscles which are most resisting, that is, the thickest. In applying this theory to the heart, we find that the thickness of the ventricles is in proportion to the weight which they have to move; and we can easily understand how a force, which would be sufficient to overcome the resistance and induce dilatation of the right ventricle, might also act as a stimulus to the left, and there produce hypertrophy: it must not, however, be thought, that when the left ventricle becomes hypertrophied, it cannot become dilated at the same time; and that, when the right is dilated, hypertrophy cannot also take place. Observation proves, on the contrary, that in the two sides of the heart, hypertrophy with dilatation is the most common form. It follows, from the preceding remarks, that we do not adopt the hypothesis of MM. Bertin and Bouillaud, which explains the greater frequency of hypertrophy in the left ventricle by the more stimulating nature of the blood that traverses it. The fact which these authors adduce, viz., that in case of the continuance of the foramen of Botal, when the arterial blood passes in a certain quantity into the right ventricle, this latter is always hypertrophied, proves nothing for their theory; for the arterial blood, which is a morbid stimulus for the right cavities, can never be otherwise than a partal stimulus for the left ones; if it were otherwise, the left auricle would be more often hypertrophied than the right auricle, whilst just the contrary happens.

1st. *Predisposing Causes.*—Among these causes, we shall place all those that tend to increase the pressure which the blood, in the healthy state, makes against the walls; such as certain malformations of the circulatory organs, as congenital narrowness of the aorta, or of the pulmonary artery; an original disproportion between the different cavities of the heart, plethora, an innate and hereditary predisposition, &c.

Exciting Causes. — 1st. *Physical Causes.* Under this head are included all those which accelerate or retard the circulation, and augment the pressure of the blood on the heart. This class comprehends all violent and prolonged efforts (causes which accelerate the circulation); numerous alterations in the aorta, the valves of the

heart, and the pericardium (as adhesions of the pericardium); certain pulmonary affections (emphysema, asthma, chronic catarrh); all abdominal diseases which cause the diaphragm to be forced upwards (the use of too tight corsets, &c.); all causes that embarrass the circulation, and increase the pressure which the blood exercises on the heart. 2d. *Physiological causes:* all moral emotions, all disorders of innervation that induce frequent palpitations. 3. *Pathological causes:* inflammation, either of the heart itself, or of its two serous envelopes. So far we have considered the development of hypertrophy as subordinate to mechanical causes only; but it is now clearly shown, that chronic inflammation, whether original or consequent on acute inflammation, may cause hypertrophy of a muscular organ; this inference may very justly be drawn as regards the heart, or central organ of the circulation. Consequently, we are forced to admit, that when an organic lesion, the consequence of inflammation, has caused hypertrophy, the inflammation itself has contributed its share to produce this alteration.

Let us now see, how the mechanical causes, which are the most frequent, act in the production of this disease; that is, in what order the different cavities of the heart become hypertrophied, when the circulation is obstructed.

General rule: every obstacle to the circulation acts at first on the cavity situated behind it. Thus, obstacles situated in the aortic system act first on the left ventricle; these add to the pressure which the arterial column exerts upon it; the ventricle has, therefore, to contend against a greater number of the causes of hypertrophy, a disease to which it is otherwise predisposed, from the greater thickness of its walls. The left auricle remains sound, whilst the ventricle completely forces out the blood which it contains, because it is protected by the auriculo-ventricular valve; but as soon as the heart has lost the power to disburden itself completely, there results an obstacle to the passage of the blood from the auricle, whose influence is extended, behind, through the lungs, to the right side of the heart, where it developes the same series of phenomena; and consequently a hypertrophy of all the cavities, or of those only which, by their structure, have the greatest tendency to this morbid transformation, takes place. When the auriculo-ventricular orifice is contracted, especially if the contraction be considerable, the left ventricle, situated in the front of the obstacle, only receiving a small quantity of blood in order to stimulate it, becomes attenuated, and sometimes softened. But the left auricle, which, at the same time that it contends against the obstacle situated before it, has to sustain the pressure of the blood which comes to it continually from the lungs, becomes hypertrophied and always dilated. This engorgement of the left auricle is not long in extending through the lungs, until it reaches the right ventricle, of which it often causes dilatation and hypertrophy. It is evident that in those cases where the pressure is made through the lungs, on the right

ventricle, the former suffer the most; for they receive constantly a quantity of blood, which is much more considerable than that of which they can disburden themselves. The deficiency of the mitral valve exerts over the lungs and right auricle a no less dangerous influence than the contraction of its corresponding orifice: for the auricle supports, in this case, all the pressure caused by the contraction of the ventricles. The right cavities also undergo the same alterations as in the preceding case.

When the obstacle to the circulation has its seat originally in the lungs, it first exercises its influence on the right ventricle, which is placed immediately behind it; then, at a later period, upon the right auricle, in the same way as it does in the left cavities. This engorgement of the right auricle forms a great impediment to the return of the venous blood from all parts of the economy; and its effects are extended through the medium of the capillaries to the arterial system, and, finally, even to the heart. It is thus that we explain how the left cavities become hypertrophied in consequence of an obstacle situated behind them in the course of the circulation, and how, for example, the left ventricle is hypertrophied, in consequence of contraction of the left auriculo-ventricular orifice.

Let us add, that the exciting causes of hypertrophy are, at the same time, those of dilatation, and that, other things being equal, it is on the proportional force of the cause relative to the reacting energy of the cavity, that the production of hypertrophy, dilatation, or of both these affections together, depends; consequently, it may be stated, as a general rule, that continued congestion causes dilatation, whilst an obstacle to the circulation, without constant engorgement of its cavity, tends to produce hypertrophy.

Hypertrophy, dilatation, and contraction, combined in different ways, in the cavities of the heart, give rise to complex affections, which vary in frequency, and of which Hope has furnished us this table, commencing with the most frequent.

FOR THE VENTRICLES.

1. Hypertrophy with dilatation of the left ventricle; same disease in the right ventricle, but less marked.
2. Hypertrophy with dilatation of the left ventricle; simple dilatation of the right ventricle.
3. Simple dilatation of both ventricles.
4. Simple hypertrophy of the left ventricle.
5. Dilatation with attenuation of the left ventricle.
6. Hypertrophy with contraction of the left ventricle.
7. Hypertrophy with contraction of the right ventricle.

FOR THE AURICLES.

1. Distension of the auricles, especially of the right, in consequence of congestion.
2. Dilatation with hypertrophy.
3. Simple hypertrophy.
4. Hypertrophy with contraction (almost unknown).

*Symptoms.** — A. *General Symptoms.*† Whilst the hypertrophy is not very considerable, and the patient keeps quiet, he experiences little or no *dyspnœa*, but this latter returns in a regular manner, and by paroxysms. In simple hypertrophy, these attacks are not of a violent character; it is only when serous congestions have taken place that dyspnœa becomes almost habitual. In general, there is little or no *cough* at the commencement of hypertrophy; it supervenes, together with external dropsy, and especially with sanguinolent congestion, but it is never, unless as a complication, very considerable. *Hæmoptysis* is sometimes, but rarely, observed. Facts have not confirmed the opinion of Bertin, who ascribes to the right ventricle, when it is hypertrophied, the same importance in the production of hæmoptysis, that the left ventricle exercises in its relation to the brain. As M. Bouillaud has remarked, the diseases of the pulmonary artery are rare, so that hypertrophy of the right ventricle is scarcely ever considerable, unless its cavity be diminished, and with it also the quantity of blood, which it forces into the lungs; finally, when the pulmonary vessels are gorged, the respiration is accelerated and becomes more active, so as to present a greater proportionate surface with which the blood shall be in relation. In hypertrophy with complication of the left ventricle, the *pulse* is almost always irregular, and bears a direct proportion in force and volume to the thickness and capacity of this ventricle. In simple hypertrophy it is stronger, fuller, and more tense than in the normal condition; it swells gradually, and forcibly; it expands largely, and it is felt for a long time under the fingers. These characters are still better marked in hypertrophy with dilatation. When, however, the dilatation has gone too far, the pulse becomes soft and compressible, though it be still full and sustained. In hypertrophy with contraction, it is tense but small; expanding slightly under the finger; and when the contraction is considerable, it is feeble at the same time that it is small. The *force*, *largeness*, and *prolonged tension of the pulse*, are the characters which denote hypertrophy of the left ventricle. It should be known, however, that in some cases of hypertrophy with dilatation, in which there is a feebleness or

* All that we are about to say here will only apply to simple hypertrophy, that is, without the complication of disease of the valves and large vessels. We shall incidently trace the symptoms of hypertrophy with dilatation, which is no more than an advanced stage of the same disease, and those of hypertrophy with contraction, which, when it is very marked, constitutes an obstacle to the circulation entirely similar to that caused by a disease of the valves.

† MM. Bertin and Bouillaud have imagined that it was wrong to refer to the hypertrophy, the serous infiltration and all that series of symptoms which indicate embarrassment of the circulation; and that simple hypertrophy without complication, is characterised by the signs of increased activity and energy of the circulation. Notwithstanding the respect which we have for the opinions of these authors, we continue to refer the preceding symptoms to hypertrophy, because we think that the same activity of the circulation which gives rise at first to active hemorrhages, produces, as an immediate effect, at a more advanced stage, engorgement of the arterial capillary system.

exhaustion of the nervous system, whatever may be the cause (advanced stage of the disease, debilitating influences), the pulse does not always present the preceding characters. The patients often experience some *affection of the head;* especially pulsations, and lancinating pains, of more or less severity, in this part; sometimes vertigo, *tinnitus aurium,* and scintillations; sometimes a lethargic somnolency, which almost entirely deprives the patient of his moral and physical faculties; sometimes, finally, in some cases, apoplexy, or an inflammation of the brain.*

The *external appearance* of the patient is not less deserving of our attention. In general, hypertrophy increases the colour of the skin. In persons naturally of a high colour, the skin assumes an extremely vivid tint; at a later period, when the capillary circulation is embarrassed, the cheeks present violet patches; the lips and the nose become purple or livid, and the adjacent parts are of a pale yellow colour. In subjects, on the contrary, whose complexion is naturally light, hypertrophy only slightly increases the general vascularity of the face, and is soon replaced by a cadaverous and yellowish colour, which sometimes extends to the lips; the eyes often participate in this general congestion; thus, they are sometimes brilliant and sparkling, and at other times strongly injected; as to the softening and loss of the eye, of which Testa has spoken of as peculiar to diseases of the heart, if it does exist, happens very rarely; and MM. Bertin and Bouillaud have ascribed it to the obliteration of the ophthalmic arteries. *Serous infiltration* supervenes at a very advanced stage of the disease; it commences most frequently in the inferior extremities, and gradually becomes general. In a certain number of cases of hypertrophy, *nervous disorders* next succeed; the patients complain of a dull pain in the præcordial region, which

* The influence which hypertrophy of the heart exercises over cerebral hemorrhage, has for a long time engaged the attention of pathologists: alternately rejected and admitted, this opinion has, at present, a large number of adherents; among them are MM. Richerand, Bertin, and Bouillaud, Bricheteau, Hope, &c. This question appears to us to be definitely settled in the affirmative, notwithstanding the labours of M. Rochoux. It must not, however, be forgotten, that the ossification of the cerebral arteries, so common in old persons, constitutes by itself alone a sufficient cause of cerebral hemorrhage. Moreover, this alteration of the vessels is generally connected with hypertrophy of the left ventricle, of which it is often the result. These two causes united may concur in facilitating the production of apoplexy. Hypertrophy with contraction never produces by itself the like results, in consequence of the small quantity of blood which this ventricle forces into the brain. It is the same with respect to all valvular obstacles, all contractions of orifices, especially that of the aortic orifice; but it is necessary that all these obstacles be considerable.

Are the hemorrhages which supervene on hypertrophy of the ventricles, and especially of the left ventricle, the result of the increased impulse of the column of blood, driven by this ventricle, or of the distension, — a mechanical engorgement of the vessels? In other words, are these hemorrhages *active* or *passive?* We think that they are only active in some very rare cases, where no obstacle exists to the circulation, and where the arterial tubes are entirely untouched; in all other cases, they are passive.

extends to the shoulder and even to the left arm, and is increased by all kinds of exercise.

Are there any general signs, by which we can tell hypertrophy of one of the ventricles in particular? According to Corvisart, more difficult respiration and a deeper colour of the face are sufficient to announce hypertrophy of the right ventricle; there is, also, another sign mentioned, viz., expectoration of arterial blood. But experience has not confirmed these opinions; and it is now proved, that the only general signs, which are of any value in this diagnosis, are — 1st, the absence of that large and strong pulse, which belongs to hypertrophy of the left ventricle; 2d, the swelling of the jugular veins, accompanied by pulsations, which are synchronous with those of the arteries. This last sign, which Lancisi had referred to aneurism, that is, to hypertrophy with dilatation of the right ventricle, is not always (as M. Bouillaud thinks), the result of regurgitation through the mitral valve; it often depends on hypertrophy with dilatation of the right ventricle, as we have shown in speaking of the general symptoms of diseases of the heart. We commonly refer to hypertrophy of the ventricle a full, tense, and vibrating pulse, flushed face, sparkling eyes, flushes of heat, vertigo, bleedings from the nose, &c. It is easy to see that these symptoms are not very characteristic. There exists no general sign, which we can make use of, to distinguish hypertrophy of the auricles from a disease having its seat in the ventricle or corresponding orifice.

B. *Local Symptoms.* — 1st. *Physiological Symptoms.* — Hypertrophy is not accompanied by pain in the præcordial region; but, when it is very considerable, the patients experience an uneasy feeling of oppression and weight; and the pulsations of the heart acquire a morbid increase in their force and frequency. Thus, we often observe violent palpitations, returning by paroxysms, under the influence of all stimulants, and fatiguing the patient, who is conscious of the force of the beats of the heart. The *impulse* presents modifications which are important to be studied; in general, it is perceived lower than natural, in consequence of the depression of the apex of the heart. In *simple hypertrophy* it is much increased in strength. The more advanced the disease, the more marked is the elevation of the thoracic parietes. If to this impulse, which corresponds to the systole, be added a diastolic impulse, it may be asserted that the disease is farther advanced. This diastolic impulse results from the diastole of the ventricles, which, during this stage of the contraction of the heart, retire from the thoracic parietes, so as to return afterwards with a kind of shock, or impulse, the result of their repletion; it forms a valuable sign in simple hypertrophy of the ventricles, and is met with also in hypertrophy with dilatation and contraction, but it never exists in the healthy state, nor even in dilatation with hypertrophy. It is rare, in simple hypertrophy and in hypertrophy with contraction, for the impulse to exceed the limits of the præcordial region. In estimating the force and extent of the impulse, in the different forms of hyper-

trophy, we must take into consideration the thickness of the walls of the chest, the condition of the patient, whether he be well made or lean, and his age. In *hypertrophy with dilatation*, the signs are made up of those of hypertrophy and of dilatation. The impulse is composed of sudden and violent shocks, which, at each contraction of the heart, cause the whole chest and the epigastric region, and sometimes even the whole body of the patient to vibrate; the diastolic impulse is at its maximum. The pulsations of the superficial arteries are often distinguishable by the naked eye. The impulse is generally felt under the whole extent of the hand; but it is not uncommon for it to extend to the summit of the chest, sometimes even behind, on the left side, in lean persons and in children. In *hypertrophy with predominance of dilatation*,* the impulse is ordinarily slight, except during the palpitations; but its characters differ widely from those which belong to the impulse of simple hypertrophy; the pulsations are strong, and produce a sound analogous to that which is caused by the blow of a hammer; but it seems to strike a small space, and is, in a measure, spent against the thoracic parietes, and does not communicate to the ear a sensation of rising proportionate to its force. These blows differ from the impulse caused by an active hypertrophy, since, in this last, the swollen ventricles seem to be in apposition, in their whole length, against the walls of the thorax, which yield to the effort: whereas, in the first case, the apex of the heart alone appears to strike these walls with a sharp blow, which is capable of producing in them a vibration rather than a real elevation.

In *hypertrophy with contraction*, the impulse is strong, but limited to a small extent of the chest. It is rare, in hypertrophy of the heart, for the beats to be irregular at the commencement of the disease; at a later period, on the contrary, when the strength of the patient is sinking, especially when death is approaching, the beats become irregular and intermittent. This irregularity is met with also, when the patient is subject to attacks of dyspnœa. The impulse of the heart diminishes under the influence of all debilitating causes, and very often it ceases entirely, or only consists of weak pulsations in subjects who are prone to violent dyspnœa, whatever be its cause. Under these circumstances, the sounds lose their clearness; consequently we should draw no conclusion, from an examination made at this time. The arching is often met with in considerable and old hypertrophies; it results from the projection of the ribs and sternum, and from the widening of the intercostal spaces.

Physical Symptoms.—Percussion.—The dulness in the præcordial region is increased in the two first varietes of hypertrophy: it is especially considerable in hypertrophy with dilatation (being 3, 4, and even 6 inches in diameter). This dulness extends most transversely and inferiorly, and it gives to the finger that percusses

* Or rather, in *dilatation with hypertrophy*.

a feeling of resistance, which is imparted neither by dilatation nor by effusions into the pericardium. In hypertrophy with contraction the dulness is never increased.

Auscultation. — Hypertrophy tends to deaden the intensity of the sounds of the heart: in *simple hypertrophy*, the first sound is dull and prolonged, and it is almost entirely extinguished when the hypertrophy is considerable: the second sound is very feeble, and in extreme cases is hardly sensible. The interval of repose, which follows the first sound, is much shorter than usual, because this is prolonged. In *hypertrophy with contraction*, the two sounds are still more feeble; and in this form of hypertrophy, as in the preceding, the first sound is scarcely audible below the left clavicle, and at the superior part of the sternum; but, in general, the second sound is heard at these points. In *hypertrophy with dilatation*, the sounds are at their maximum; they are more distinct, than in any other disease of the heart. The first sound derives from the dilatation its clear and sudden commencement, and from the hypertrophy its prolonged termination; as to the second sound, it does not change its natural tone; it is only clearer than in the normal state. These sounds are often perceived, in the whole extent of the chest, both before and behind, in lean persons and in children. In a very small number of cases of hypertrophy with dilatation, we find, on a line with the orifice of the aorta, a slight murmur, which is soft and feeble, accompanying the first sound, and which, according to Hope, results from the anemic state of the persons affected. In *hypertrophy with predominance of dilatation*, the first sound is not as clear as in the preceding variety: it has not that prolonged termination, but is short and sudden, like the second: as to the second sound, it is not altered, but is a little clearer than usual. In fine, in all forms of hypertrophy, the sounds may be enfeebled by the same causes that enfeeble the impulse, and of which we have already spoken.

The *local symptoms* which characterize hypertrophy of each one of the ventricles, in particular, are the following: in that of the *left ventricle*, the impulse is principally felt in the region of the cartilages of the fifth, sixth, seventh, and even eighth ribs; the dulness, as well as the arching, extends principally in this direction: in the disease of the *right ventricle*, the impulse is principally perceptible under the sternum or in the epigastric region; the dulness and the prominence are especially perceptible at the sternum. There exists no other local symptom which will serve to point out general hypertrophy of the auricles, or hypertrophy of each one of them in particular.

Diagnosis. — Hypertrophy of the heart is distinguished, 1st, from *pericarditis*, by the nature of the dulness, the force and energy of the impulse, the absence of the friction sounds, and the reactional phenomena which belong to this disease; 2d, from *endocarditis*, by the increased extent of dulness, absence of the murmurs, and sympathetic phenomena, which accompany this disease; 3d, from *pleurisy*

with effusion, which sometimes forces the heart under the sternum, by signs which announce this alteration in the seat of the organ, and the absence of that dulness in the posterior part of the thorax which belongs to pleurisy; 4th, from *pulmonary emphysema*, by the normal resonance of the whole chest, and dulness in the præcordial region. (See, farther on, the *diagnosis of this last disease, and of dilatation with attenuation from diseases of the valves, and of aneurisms of the aorta.*)

Progress, terminations. — The progress of hypertrophy is generally slow and gradual; sometimes, however, it is rapid. So long as the hypertrophy is inconsiderable and uncomplicated, it has little influence over the general health: the patients who are affected with it may live for a number of years, if they are temperate in their habits and diet; they only experience a little difficulty of respiration and slight palpitations. But if these individuals indulge in excesses, or are occupied with fatiguing labours, the disease makes rapid progress; and they fall victims to apoplexy, or to an aggravation of the original disease. The rapidity of this fatal termination is subordinate to many circumstances; to the form of the disease (hypertrophy with dilatation is the most dangerous of all); to its complications (contractions of the orifices, diseases of the aorta or pulmonary artery, febrile diseases, adhesions of the pericardium); to the persistence of the exciting causes of the disease, and to the strength of constitution of the patient.

Prognosis. — Favourable at the beginning of the disease, it becomes grave at a more advanced period, especially when the dropsy cannot be controlled; but we must note the conditions in which the patient is placed, especially the complications. We can generally, by proper treatment, succeed in radically curing a simple hypertrophy, provided the person affected be of a good constitution, and has not passed the age of forty years: the chances of cure diminish after this age; and at fifty or fifty-five, it becomes very difficult.

Treatment. — The first indication which is presented in hypertrophy, is to remove the known exciting causes, as corporeal exercise, intemperance, moral emotions, &c.; then it will be proper to have recourse to a measure (*sanguineous depletion*), which has been always recommended, but differently employed, in the treatment of this disease. Laennec, after the example of Albertini and Valsalva, suggested very large bleedings, at intervals of two, four, or eight days; and a reduction gradually of the quantity of nourishment to fourteen ounces a day. This treatment should be continued for many months, at least; sometimes for several consecutive years. The end proposed by Laennec, is to unload the cavities of the heart, and to diminish the stimulating action of the blood and the active nutrition of this organ. The treatment of Albertini, applied rigorously, often counteracts its intention, and may hasten the death of the patient. We think that an equally fortunate result may be obtained by a mode which is more advantageous to

the patient, — which is, to draw six or eight ounces of blood, by venesection, every fifteen days or a month, according to the age or strength of the subject, and afterwards to have recourse to local bleedings, behind the ears, or over the præcordial region, if there be symptoms of cerebral or cardiac congestion. The nourishment should consist, during the first two or three months, of fresh fish, vegetables, and farinaceous articles (only feeble persons may eat a little meat); the nourishment should be small in quantity, and the meals light, and so arranged as to satisfy the appetite, without overloading the stomach. The patient should have water, slightly flavoured, for drink: those who are feeble may drink a little wine largely diluted. All kinds of exercise on foot should be prohibited; but riding in a carriage will be found useful. It will be well, during the first period of the disease, to join, to the use of bleedings, saline purgatives with the view of preventing constipation, and of producing a slight revulsion on the intestinal canal; the use of nitrate of potassa, digitalis, and other diuretics, will be indicated, even when dropsy has not appeared. Digitalis, independently of its diuretic action, has the property of rendering the beats of the heart slower, and of considerably diminishing their force: given in the dose of one grain to four grains of the leaves, or of ten to thirty drops of the tincture, it generally produces good effects. It is especially useful in simple hypertrophy, less so in hypertrophy with dilatation, and would be very pernicious when the dilatation has reached to a great extent. This medicine ought not to be continued during the whole period of the disease, as some persons practice. When we have obtained the effects which we expected, it is proper to suspend its use, in order to recur to it at a later period with advantage: if the digitalis be constantly employed, it will produce disorders of the digestive canal; cardialgia, nausea, vomiting, &c. There are some persons whose stomachs cannot tolerate it at all; in this case it should be used by enema, or the endermic method should be tried.

When the dropsy is perfectly well marked, we have recourse to the most active diuretics, and to hydrogogues. (See *Treatment of the Diseases of the Valves.*)

Under certain circumstances, where the irritation of the nervous system continues, notwithstanding the most judicious measures, it will be found useful to administer some sedatives, as hyosciamus, one grain to four grains of the extract, the acetate or hydrochlorate of morphia in doses of from a tenth to a fifth of a grain, &c.

The employment of iodine and mercurial frictions, as absorbents, has been proposed; but the results have not answered expectation.

The treatment which we have just proposed, and which is very similar to that which Hope employed in his large practice, ought, in order to succeed, to be perseveringly continued for two or three years: and it will be necessary, for one or two years more, to take proper precautions in order to avoid relapses; but, although the treatment be thus prolonged, the patients easily submit to it, since it does not give them much trouble or distress.

CHAPTER II.

ATROPHY OF THE HEART.

Atrophy, the name given to a defect of nutrition, is characterised by a sensible diminution of the size and weight of the heart: it presents three varieties:

1. *Simple atrophy;* attenuation of the walls, without notable change in their capacities, but with an appreciable diminution in size.

2. *Atrophy with dilatation;* walls of one or more cavities attenuated; capacity increased in extent, size slightly larger, but weight diminished.

3. *Atrophy with contraction;* thickness of the walls equal, or even exceeding that of the normal state, but size of the cavities diminished.

Atrophy is general or partial; *general atrophy*, especially atrophy with contraction, may deprive the heart of a third, and even half its weight and natural size. Thus, Burns found, in an adult, a heart which did not exceed in size that of a new-born infant; and in a girl, twenty-six years old, one which appeared to belong to a child six years old. In the case where the heart loses at the same time its volume and size, it often presents a shrivelled, dried up appearance, which Laennec has compared to that of a *rotten apple.*

The causes of atrophy are referrible to the heart, to its membranes, and especially to a particular state of the constitution. The atrophy may follow a considerable hypertrophy, contraction of the coronary vessels, the compression caused by an effusion into the pericardium or large pseudo-membranous laminæ; but, most generally, it is the result of chronic diseases, which alter the qualities of the blood, as chlorotic, scorbutic cachexies, &c.; finally, it has been seen to supervene on the (depleting) treatment of Albertini.

Atrophy of the heart is characterized by signs, which are entirely opposite to those of hypertrophy.

Local Symptoms. — Beats of the heart, short and feeble; impulse hardly appreciable; sensible diminution in the dulness on percussion; extreme feebleness of the sounds of the heart, which are slow and regular.

General Symptoms. — Atrophy often exists for a long time, without being accompanied by general symptoms; individuals, who are affected with it, are predisposed to anemia, to syncope, and to other nervous affections. In some cases, however, we observe shortness of breath on the least exertion, palpitations without energetic contractions, discoloration of the integuments, and a precarious and sickly existence, œdema, and death, in a state of exhaustion.

The treatment of atrophy is governed by the exciting causes.

CHAPTER III.

DILATATION OF THE HEART.

Dilatation of the heart is a disease which consists in an increase of the capacity of one or more cavities of this organ. In this affection, the walls of the heart may be thickened, or in the natural state, or much attenuated. Hence we have three varieties of dilatation, corresponding to these three states of the heart:

1. *Dilatation with hypertrophy.** — In this variety the cavity is enlarged, and the walls acquire an anomalous thickness.

2. *Simple dilatation.* — The cavity is enlarged, and the walls preserve their natural thickness.

3. *Dilatation with attenuation.* — The cavity is enlarged, and the walls lose their thickness.

We sometimes find two and even three forms of dilatation in different points of the same cavity.

Anatomical Characters. — Sometimes the muscular substance of the heart preserves its soundness; but, most generally, the walls are slightly flaccid and even softened. Their colour may be more deep or more pale than in the normal state.

In speaking of hypertrophy, we have mentioned the first and second varieties; it now remains to speak of the third, or *dilatation with attenuation.* It is rare for this affection to be confined to only one ventricle; but it is always more marked in the right than in the left ventricle. The attenuation may be so great as only to allow two lines in its greatest thickness to the left ventricle, and to reduce its apex to a simple membrane of scarcely half a line in thickness.

The *columnæ carneæ* are more separated from each other than in the normal state; in general, the attenuation is always less marked in the inter-ventricular septum. As dilatation takes place more in the transverse than in the longitudinal direction of the ventricles, the heart assumes a spherical form, and its apex is hardly perceptible (*cœur en gibecière*, Laennec). When the auricle and corresponding ventricle are much dilated, it may happen that the orifice which is common to them will become also dilated, and the valves be incapable of closing it; this is one cause of regurgitation, and

* Of these varieties the first is identical in its nature with that variety of hypertrophy called *hypertrophy with dilatation;* the only difference is in degree; and the position of the word *dilatation* indicates that the difference is in favour of this last affection. The second variety is equally identical with this variety of hypertrophy which we have called *hypertrophy by increase in the extent of the walls* without change in their thickness. When the dilatation is extreme, this variety acquires the name of *simple dilatation.*

is an alteration which can only be recognised by examination after death.*

When the dilatation is very extensive, especially where it is accompanied by softening of the muscular substance, a rupture may be produced, and death take place suddenly, in consequence of the effusion of blood into the pericardium.

The auricles, protected by the auriculo-ventricular valves, are much less exposed to dilatation than the ventricles. As they are much oftener distended than dilated by the blood, it is necessary to know the normal relation which exists between the different cavities of the heart, and to have the means of distinguishing dilatation from distension of these cavities. "The cavities of the heart," says Laennec, "are very nearly equal, as regards each other; but as the parietes of the auricles are very thin, and those of the ventricles have much thickness, the first only form one-third of the whole size of the organ; or what amounts to the same thing, the size of the auricles is about half that of the ventricles." Let us add, that the right auricle is generally distended, that it is much longer and more flattened, which makes it appear larger without its being so in fact. The method of ascertaining whether dilatation of the auricle exists or not, is due also to Laennec. According to this author, in distension, the walls of the auricle are strongly contracted on the blood which they contain, and the thinnest parts allow its colour to be perceived. A dilated auricle has not the same tense appearance, and its walls are more opaque. When we force the blood into the vessels without opening its cavities, these last, if they are simply distended, resume nearly their natural size; whereas, when they are dilated, they preserve almost the same which they had when they were full. Moreover, it is not common for the auricles to be dilated without their parietes undergoing a certain degree of thickening. This method, which Laennec employed in order to distinguish the distension of the auricles from their dilatation, is equally applicable to the ventricles. In short, when the blood is pressed from the ventricle into the auricle, if the former be dilated, it preserves its enlargement, and its walls are always more or less flaccid; whereas, if it is only distended, it resumes in some measure its normal size, and its walls still retain a certain degree of firmness.

Causes. — Every time that the blood, accumulated in the cavities

* Subjoined is the measurement of the orifices, in their natural and dilated state, as laid down by M. Bouillaud.

		Medium.		Maximum.		Minimum.	
		in.	lines.	in.	lines.	in.	lines.
Aortic orifice	Natural	2	5½	2	8	2	4
	Dilated	,,	,,	3	5	,,	,,
Pulmonary orifice	Natural	2	7¾	2	10	2	6
	Dilated	3	2⅗	,,	,,	,,	,,
Left auriculo-ventricular orifice	Natural	3	6	3	10	3	3
	Dilated	4	1½	,,	,,	,,	,,
Right auriculo-ventricular orifice	Natural	3	10	4		3	9
	Dilated	5	2	5	9	4	2

of the heart, exerts a considerable distensive pressure against its parietes, and overcomes the resistance which their contractile and elastic force oppose to it, a dilatation is the result. Now, as this power of resistance is in proportion to the thickness of the walls of these cavities, it thence follows, that, everything else being equal, the cavities which have the thinnest walls are the most susceptible of dilatation; and this conclusion holds good in relation to the right ventricle and the arteries. In order that the dilatation should become permanent, it is necessary either that the exciting cause be prolonged for a certain length of time, or that it be repeated at short intervals.

The causes of dilatation are, — 1st, a conformation of the heart whose contractile power is not in relation with the rest of the economy; 2d, all obstacles to the circulation which have their seat at the orifices of the heart, or in the aortic or pulmonary system; 3d, all causes which diminish the resistance of the walls of the heart (inflammation of the organ or of its membranes, rheumatism and gout). The dilatation of the auricles is generally caused by a disease of the corresponding auriculo-ventricular orifice; although, under some circumstances, as where the valves are sound, and where the ventricle only partially rids itself of its contained blood, the stagnation of this fluid acts in proportion to the distension of the auricles, in the same manner as in the case of the disease of the auriculo-ventricular orifice. Let it be observed, however, that, for dilatation to be produced in this exceptional case, it is necessary that this action be permanent, or at least prolonged, which rarely happens.

Symptoms. — A. *General Symptoms.** These are, *serous* infiltra-

* Notwithstanding the opinion of some pathologists who regard dilatation as always consecutive to another lesion, we shall continue to refer all the general symptoms which indicate embarrassment of the circulation to *primitive* dilatation. In the normal state, there exists a certain relation between the weight of the column of blood which the heart is obliged to move, and the power of the heart itself; if, in consequence of some causes (among which diseases of the heart and arteries may be certainly counted), the weight with which this column presses on the heart is more considerable than this organ can naturally support, dilatation will result. But is it not also true, that this increase of pressure may depend not only on an increase of the weight of the circulation, but, also, on a deficiency of power, or the relative debility of the heart, whether it be congenital or acquired? Dilatation, when it is present in these last circumstances, should be ranked among primitive diseases with as much propriety as hypertrophy; and even when it coincides with mechanical obstacles, it is impossible not to attribute to it a large proportion of the symptoms which indicate embarrassment of the circulation; in proof of which, we find that the patients can bear without much inconvenience an organic disease of the valves or of the aorta, as long as there is no hypertrophy, nor dilatation, nor softening of the heart exists; but as soon as these last affections supervene, the symptoms assume a more serious form, and the disease rapidly progresses to a fatal termination. It remains to know in what manner dilatation produces all these phenomena of disordered circulation. By dilatation, the muscular fibres of the heart are placed in a greater state of tension than usual, whilst, at the same time, their contractile power diminishes; they lose in force what they gain in length. It is this deficiency of power which con-

tion, which is first seen in the extremities, then in the serous cavities, and is coincident with the œdema of the lungs; a *peculiar colour of the integuments*, especially of those of the face, which becomes purple, or violet, sometimes of a leaden colour or livid, with tendency to coldness of the extremities; *cerebral congestion*, which takes place a few days before the fatal termination; *cough*, sometimes associated with expectoration; great *dyspnœa*, returning by paroxysms, especially in the night; a *pulse*, soft and feeble, often irregular, intermittent, and unequal, when softening is joined to dilatation; *passive hemorrhages*, which take place either from the nose, the stomach, the intestines, or the uterus, and, more rarely, from the bladder; *swelling and congestion of the liver*, which is a consequence of the engorgement of the right side of the heart, and which induces ascites, icterus, hematemesis, intestinal hemorrhage, hemorrhoids, and, indirectly, uterine hemorrhage; *angina pectoris*. These are the signs of dilatation of the ventricles It remains to ascertain which ventricular cavity is affected with dilatation. According to Corvisart, the *most certain general signs of dilatation of the right ventricle* are, more considerable dyspnœa than in the affections of the left, a better marked serous diathesis, more frequent hemoptyses, and a more evident lividity of the face, and even a very deep violet colour.

There is no doubt that these symptoms belong to dilatation of the right ventricle; but they no more belong to this affection than to hypertrophy with dilatation of the same cavity, and to certain diseases of the valves of the left side of the heart. We believe, in common with Laennec, that the most constant and the most characteristic of the general signs of dilatation of the right cavities, is the *permanent distension of the external jugular veins, without sensible pulsations*, a distension which does not disappear even when we compress the veins in the superior portion of the neck, and prevent the afflux of blood to them.

As to *dilatation of the auricles*, it does not present other general symptoms than those which depend on the disease of the ventricle, or corresponding orifice.

B. *Local Symptoms.* — 1. *Physiological Symptoms.* — *Palpitations*, not violent, but returning quickly under the influence of the least moral stimulus. The *impulse* loses its force, sometimes is entirely wanting: when it is perceived, it consists in a short percussion of the thoracic parietes, which does not raise the ear applied to it: if the dilatation be considerable, the impulse is perceived a little lower than in the normal state.

Sometimes, of many pulsations of the heart that *are heard*, *one*

stitutes the obstacle to the circulation, in the same manner, if we may be allowed the comparison, that the movements of a watch are retarded by the feebleness of the main-spring (Hope). None of these remarks are applicaable to dilatation with hypertrophy, in which the activity of the circulation is increased instead of being diminished, even when the hypertrophy is very slightly marked.

only is felt, and when this last is strong, Hope concludes that the walls are not very thin. When the impulse is felt at the inferior part of the sternum, we may *suspect dilatation of the right ventricle.*

2. *Physical Symptoms.* — *Percussion.* The resonance of the præcordial region is diminished; the dulness descends a little lower than in the normal state; when perceived under the inferior part of the sternum, it indicates dilatation of the right ventricle. *Auscultation:* when the ventricular cavities are thin, without being dilated, the first sound of the heart is stronger and clearer than in the normal state, and resembles the flapping of the second sound; but, when dilatation exists, even though the attenuation has not gone far, the first acquires almost all the characters of the second sound. Finally, when the dilatation is extreme, we can distinguish the two sounds, not by their nature or intensity, but only by their maximum point of intensity, and their coincidence or want of synchronism with the arterial pulse. Moreover, in dilatation, the second sound is itself reinforced. When this disease is accompanied by dilatation of one of the orifices, if the corresponding valve does not accommodate itself to this movement of dilatation, and does not enlarge in a proportional manner, a soft murmur from regurgitation, whose intensity varies with the orifice affected, is heard. In proportion as these two sounds increase in intensity, they are perceptible over a greater extent of the chest. According to Laennec, when the sounds of the heart are audible below the ordinary limits, they are heard successively in the following regions: —

1st. Along the sternum, and the left antero-posterior side of the chest as far as the clavicle.

2d. Over the same space in the right side.

3d. The left lateral portion of the chest, from the axilla to the corresponding region of the stomach.

4th. The right lateral portion over the same extent.

5th. The left posterior portion of the chest.

The intensity of the sound diminishes progressively in the order of succession which we have just indicated, provided that the parts which surround the heart be in the same relative state. This scale of dilatation is not perfect, but it is much preferable to that by Dr. Hope, which consists in the investigation of the difference that exists between the first and second sounds, and in what points they resemble each other; and in a comparison of the intensity of the first sound *with what we know by experience to be the natural condition in a like subject.*

Diagnosis. — Dilatation of the heart is distinguished from *pericarditis* by the nature and smaller extent of the dulness on percussion, by the great *clearness* of the sounds of the heart, and by the absence of the friction sounds and of all those symptoms of reaction which belong to pericarditis; it would, also, be difficult to confound it with *endocarditis*, because, in this last disease, the dulness does not extend far; since the sounds, at first strong, are not long in be-

coming altered by murmurs; and, besides, there is a febrile state which is not found in dilatation. Finally, it can be distinguished from *hypertrophy* by means of the following signs: impulse feeble, scarcely sensible, sounds of the heart clear and audible over a large extent of the chest, considerable feebleness of pulse, tendency to venous congestions and to passive hemorrhages.

Progress, termination, prognosis. — It is not uncommon to find individuals whose heart, though sound and not dilated, has naturally thin walls. This state is marked by signs analogous to those of dilatation. Those persons who are generally feeble and delicate, may live for a great number of years in a tolerably comfortable state of health; but in febrile diseases, and in diseases of the respiratory organs, they are much more subject to dyspnœa. When the dilatation is slight, it does not constitute a very formidable disease; sometimes the patients affected with it pay little or no attention to the dyspnœa which it occasions; they only say that their health is delicate, that they have short breath and sometimes palpitations. If the dilatation, on the contrary, have reached a certain degree, and so far as to induce a morbid dyspnœa, the disease has a marked tendency to increase, unless the circulation be maintained in a state of complete repose. We may consider the tendency of dropsy to be reproduced immediately after its disappearance under proper treatment, as a fatal sign.

To recapitulate: the general prognosis is not unfavourable as regards life; as to the special prognosis, it depends on the violence of the symptoms, the constitution of the patient, and the form of the disease. (Dilatation with attenuation, and especially dilatation with softening, constitute the most dangerous forms.)

Treatment. — To render to the attenuated walls their natural thickness; to renew their elasticity so as to permit them to regain their form, and when this is not possible, to oppose the progress of the dilatation, are the indications which present themselves in the treatment of this disease. We must commence by removing the causes of the dilatation; if these be found in diseases of the respiratory apparatus, or in deleterious habits or occupations, we must, first of all, remove the former, and induce the patient to give up the latter. Unfortunately, the causes of dilatation are not all transient: and those which are permanent, not being removable so as to admit of our obtaining a cure, we must have recourse to a palliative treatment. In either case, it is necessary to give the patient moderate nourishment, and, at the same time, to keep him quiet; to procure for him a pure and dry air, riding in a carriage, and dry frictions: cold baths are then applicable, provided there be no pulmonary congestion; strong revulsives (among others, cauteries over the region of the heart), irritating pediluvia, and maniluvia, are not less useful. It is a rule, in dilatation, not to have recourse to bleedings, digitalis, narcotics — means which cause a diminution in the number and energy of the contractions of the heart, and produce a stasis of the blood. We are, however, some-

times obliged to employ them, but then it must be done cautiously; thus, bleedings of about six ounces from a small aperture, and only during the intervals of the paroxysms of dyspnœa, should be practised; opiates may be prescribed when the patients are subject to sleeplessness, and their rest disturbed by attacks of dyspnœa; digitalis will be useful when there are obstinate palpitations, which cannot be otherwise restrained. But all these measures should be employed with the greatest discretion, and only from the necessity of the case; and they should be abandoned as soon as the desired effect is obtained.

In the paroxysms of dyspnœa, hot maniluvia and pediluvia will be found very useful: the patient should be well covered, but yet, the access of fresh air should be allowed; a drink composed of ether, laudanum, camphor, and assafœtida, should be given in tea-spoonful doses. As soon as perspiration takes place, the paroxysms disappear, as it were, by enchantment.

The principal design of the treatment of dilatation is to alter the whole economy; and hence, the necessity of continuing it for a long time until the patient finds himself evidently relieved; for relapses are dangerous, and we are not always able to arrest their progress.

CHAPTER IV.

PARTIAL DILATATION OR TRUE ANEURISM OF THE HEART.

True aneurism of the heart is characterised by the development of a pouch of varying size in the substance of the organ, communicating with the ventricle or auricle by a more or less narrow aperture. It is a very rare disease.

Anatomical Characters. — This affection is presented under two different forms; either there is no deformity externally to the heart, or there is a visible tumour external to this organ. All parts of the left ventricle may be the seat of this partial dilatation: it may be found at the apex, on different parts of the base, in the intermediate portions of the lateral wall, or in the interventricular septum. Its size varies from that of a walnut to that of the heart itself. In general, there only exists one aneurism, though there may be even four in an incipient state. In certain cases, numerous aneurisms communicate with each other; the three coats of the heart sometimes enter into the composition of this aneurismal sac. The endocardium which often contracts adhesions with the clots that fill the cavity of the aneurism, always increases in thickness and becomes opaque; at a later period it undergoes the steatomatous, cartilaginous, or osseous degeneration; it is lacerated, and its remains are found on the borders of the aneurismal aperture. The muscular substance

becomes thin and very pale. The cardiac fold of the pericardium loses its transparence, and is covered with white spots or distinctly false membranes. The sac is formed, in some circumstances, only by the muscular fibres, at other times only the endocardium and pericardium are found, the muscular coat having disappeared. The orifice by which the aneurism opens into the cavity of the heart is sometimes as large as, or larger than, the diameter of the tumour itself; sometimes it is narrow, in reference to this diameter; its borders are sometimes blended with the substance of the heart; at other times, they are circumscribed, projecting, and formed of a more or less dense but smooth fibrous tissue; finally, these borders may present traces of the rents of the internal membrane, and at the same time various degenerations. When the orifice of the sac is narrow, the aneurismal cavity contains clots, which are occasionally arranged in concentric laminæ, as in aneurism of the arteries; when the opening is wide, the clots are amorphous; in other cases, we find globular, yellowish clots, thick fibrinous layers separated by a sanious or purulent liquid, or saline and calcareous concretions. The aneurismal sac is generally strengthened by partial, and under some circumstances, general adhesions, which it contracts with the pericardium. This latter may also present false membranes, and serous or sanguineous effusion, &c. Besides the alterations which properly belong to dilatation, we also find the heart otherwise changed by morbid structure; most generally this organ is the seat of hypertrophy with dilatation. It is excessively rare that there is no alteration.

An aneurismal dilatation is sometimes found in the auricle. This affection, which is also more rare in this cavity than in the ventricles, is almost always diffused, and is principally seen in the sinus of the auricle. In a case mentioned by M. Thurnam, the aneurism was circumscribed: there was a sac of the size of a walnut, which rested on the base of the left ventricle, and which contained thick fibrinous concretions and liquid blood. Partial dilatation of the auricle or of its appendix always coincides with considerable contraction of the left auriculo-ventricular orifice.

Causes. — Partial dilatation of the heart is never seen in infancy; it is most frequently observed in persons between twenty and thirty, or between forty and fifty years of age. Men are more liable to it than women. Acute articular rheumatism appears to be intimately connected with the production of this disease (Thurnam).

Exciting Causes. — Whatever may be the cause which predisposes the heart to partial dilatation, this latter, together with its accompanying phenomena, appears subsequent to all the causes which give great activity to the circulation, or which increase the pressure of the blood against the walls of the ventricle; such as anger, excesses of all kinds, external violence, prolonged suspension of respiration, &c.

The *symptoms* and *diagnosis* of partial dilatation are still shrouded in great obscurity; aneurism is not always accompanied, at its

commencement, by well-marked symptoms; its progress is insidious, and it is only gradually that symptoms of organic disease of the heart make their appearance. Sometimes the invasion is sudden, and is announced by symptoms which characterise rupture of the heart, when this last is not immediately fatal; viz., acute pain in the præcordial region, orthopnœa, anxiety, fear of death, disposition to syncope, pulse vibrating and frequent but laboured. These symptoms are soon followed by others which belong to the organic diseases of the heart; cough, pulsations of the carotid arteries and of the jugular veins, livid or violet colour of the face, hemorrhage from the nose or lungs, &c.

The *local symptoms* have hitherto been noticed in too imperfect a manner, to allow us to deduce anything useful for their diagnosis. We find, in some authors, that the impulse was increased or diminished, that a bellows or rasping sound was audible; but we do not learn determinately what is the maximum of these murmurs, nor their synchronism with one or the other sound of the heart; and yet these are the most important points in diagnosis.

In four cases observed by M. Gendrin, there was heard a dry, whistling, and sibilant sound, coinciding with the systole of the heart, which ceased immediately after the systolic impulse; and after a short interval of silence there was heard a diastolic murmur, which was very short, sibilant, dry, and rough; this double murmur, at separate intervals, may hereafter be turned to account in the diagnosis of this disease, when more numerous facts shall allow of our establishing it as a constant sign.

In the present state of the science, it is more than probable that true aneurisms of the heart will be confounded with other affections of this organ, and particularly with hypertrophy. This error will be the more easy, since we always regard, as the one most probably present, the affection which is most often seen.

Progress, duration, termination. — Partial dilatation of the heart may last for a series of years without very sensibly affecting the health of the patient; its progress is extremely slow. It may terminate by rupture, which is most rare; more frequently the aneurismal cavity is filled with discoloured, plastic, and adherent clots, and the disease is prolonged in an indefinite manner.

We have nothing to say on the treatment of this disease; — it is the same with that which is employed against general dilatation of the heart, and aneurisms of the aorta.

CHAPTER V.

SOFTENING OF THE HEART.

Softening of the heart presents the following anatomical characters; the organ collapses, and becomes flattened when placed upon a table; its ventricles collapse even though they may be hypertrophied; the muscular portion is flaccid, and bends with the greatest facility; it is soft and fragile; it tears sometimes under the simple pressure of the fingers. These are the characters common to all the varieties of softening. As this disease is almost always accompanied by a change of colour, Laennec has made this conversion the basis of three varieties:— 1st. *Red softening;* in this variety the muscular substance is of a brownish-red, and sometimes violet colour; at other times it is spotted, as if there was extravasated blood; the inter-muscular cellular tissue is injected, and we find, between the muscular interstices, or below the serous membrane which either lines or invests the heart, blood more or less altered. In some cases, in the middle of the fleshy tissue of the heart, which is strongly coloured red, we meet with little blackish points, formed by fluid black blood, of the colour of wine lees (*apoplectiform softening* of M. Cruveilhier). 2d. *White softening* was shown by M. Laennec to accompany pericarditis exclusively. According to this author, this variety is accompanied by a whitish paleness of the surface of the heart, without friability; the muscular substance is slightly flaccid, and the walls of the ventricle collapse after incisions. According to M. Bouillaud, this softening is identical with that which Corvisart observed in a case of pericarditis with effusion, and which was characterised by a very marked discoloration of the walls of the heart, an evident diminution of tenacity in the fleshy fibres, and a considerable development of the vascular system of the organ. 3d. *Yellow, or yellowish softening*, is much more common than the *red*, or *white* variety; it is accompanied by a pale yellow, or fawn colour, which Laennec compared to that of the palest dead leaves; it may occupy the whole, or a portion of the heart; often it only penetrates to a certain depth of the muscular substance, below the inflamed pericardium, or endocardium.

Akenside has described, under the name of *gelatiniform softening*, a pathological state of the heart, which consists in a softening of the fibres, with friability and the infiltration of a gelatinous serosity (*senile softening* of M. Blaud).

All these kinds of softening may be either partial or general.

Most generally, when the softening has lasted a certain time, we find a dilatation of the cavities of the heart, an inevitable result of the loss of cohesion and elasticity of the walls of this organ.

What is the cause and nature of softening? According to Laennec,

it is an affection *sui generis*, produced by disordered nutrition, in consequence of which the solid elements diminish in proportion as the liquid or semi-liquid elements increase; and the change takes place without any sign of inflammation. In the opinion of MM. Bertin and Bouillaud, the softening of the heart, whatever may be its colour, is the result of inflammation. It is more than probable that the truth lies between these two opinions. Let us observe under what circumstances the different varieties of softening are met with; the *red softening* may be without doubt the result of an acute inflammation; and how can it be doubted, when we find it joined with sanguineous and purulent infiltration of the fibres of the heart? Is it not most frequently met with in the case of subacute pericarditis or endocarditis, and is it not then exactly limited to the most inflamed points? Red softening may then be inflammatory. But when, on the contrary, it supervenes after a disease which has for a long time occasioned disorder of the venous circulation, or in diseases which produce great alteration of the blood, as scurvy and typhoid fever, we are forced to acknowledge that red softening is no longer the product of a carditis, and that, in fine, it is not inflammatory.

The *whitish* softening, as will have been recognised by the description which we have given of it above, evidently belongs to an advanced stage of acute carditis with effusion of pus and of plastic lymph between the fibres of the heart. (This state of the heart is rare, since neither M. Bouillaud nor Hope have seen it.) The *whitish* softening is then inflammatory. But, as in some cases of atrophy of the heart accompanied by anemia, the walls of this organ are pale and flaccid, may we not place these two under the same head, and admit another species of white softening which is not of an inflammatory nature.

We cannot doubt that *yellow* softening may be the result of inflammation, when we see it penetrate to two or three lines in depth below the inflamed pericardium, or below the serous membrane of the heart, presenting also remains of endocarditis. But when it is met with in cachectic persons, without there having been, at a former period, any inflammation or fever which could account for this state, and especially when it coincides with a pale yellow colour of the face, puckering of the skin, and other signs of anemia, we are forced to admit *yellowish* softening from a non-inflammatory cause.

Gelatiniform softening may be regarded as inflammatory under certain circumstances, where we find traces of inflammation of the pericardium or endocardium; but when we meet it in persons exhausted by old age, without any trace of inflammation, we cannot admit its inflammatory origin.

From what precedes, it follows that the *red*, *yellow*, and *gelatiniform*, perhaps also the *white* softening, may occur independently of inflammation.

Symptoms. — A. *General Symptoms.* — When softening is the result of an acute inflammation, it almost always coincides with

pericarditis or endocarditis. The general symptoms of these two affections are so combined that it is difficult to distinguish them from each other. The pulse is active and trembling, anxiety considerable; finally, we observe all those acute symptoms which announce pericarditis with an abundant effusion, or an endocarditis with the formation of polypi in the heart.

When softening succeeds a chronic inflammation, or the action of a debilitating cause, the general symptoms are not less doubtful. We observe a yellow exsanguineous hue, or a purple, livid colour of the cheeks and lips; the pulse is frequent and weak, often irregular, unequal, intermittent; and there is a general languor, or gradual diminution of strength, with partial or, sometimes, general infiltration.

Softening often supervenes on organic diseases of the heart, especially in hypertrophy with dilatation. Laennec thinks that, when, in a patient affected with dilatation or hypertrophy, there are long and frequent attacks of suffocation and impending death, when the violet hue of the face, extremities and other points of the surface of the body has indicated, a long time before death, the stasis of the venous blood, we may admit, as probable, the existence of softening.

Local Symptoms. — The *impulse* is always more or less weak, and it often happens that the pulsations of the heart are not only intermittent and irregular, but, also, of unequal force. When, in the midst of these very feeble pulsations, we perceive one much stronger than in the healthy state, this sign is sufficient to diagnosticate hypertrophy coinciding with softening (Hope). *Percussion* shows that the heart has not increased in size, unless there are complications. By *auscultation* we find that the two sounds are feeble, and that the first becomes short and flapping like the second; there is no trace of murmur.

Diagnosis.— A group of violent symptoms indicating disorder of the circulation, among the first of which we must place irregularity of the pulsations of the heart and pulse; considerable feebleness of the two sounds, and the short and flapping character of the first sound. Such are the signs which may serve to diagnosticate softening of the heart. In its general features, there are few diseases which present so much analogy with softening as those of the mitral valve: it is, in fact, in them that the symptoms of disordered circulation, and, in particular, the irregularity of the pulse, are at their maximum. The diagnosis is very difficult. If there be no murmur at the orifices of the heart, if the pulse be irregular, and if this irregularity continue for some time, and be neither due to a nervous condition, nor to an attack of dyspnœa, there can be no doubt respecting the existence of softening. We have already said, that softening may be joined to hypertrophy of the heart; hypertrophy and softening each contributing its influence to enfeeble the sounds of the organ, it is difficult to tell to which of these two affections the symptoms are referrible. We, however, can make use of, with advantage,

the flapping character of the first sound, (never met with in hypertrophy,) as well as of all the signs peculiar to hypertrophy; increase of impulse, dulness, on percussion, more extensive than in the healthy state, &c.: irregularity of the pulse, when it exists in this particular case, adds much to the certainty of the diagnosis, because this sign does not belong to simple hypertrophy.

Progress, duration, terminations. — The progress and duration of softening varies according to a number of circumstances, and principally according to the conditions in which the persons affected are found, morbid complications, &c.

Prognosis. — This disease is rarely idiopathic, and its prognosis is always deduced from the affection which produced it: in pericarditis and endocarditis, the presence of softening adds much to the danger of the disease; but when these diseases terminate favourably, everything induces us to believe that the muscular substance returns to its normal consistence. In chronic diseases, and especially organic ones of the heart, softening is one of the most serious affections, not only in consequence of the debility of the heart, to which it gives rise, but, also, because it opposes the return of the organ to its normal proportions. We may, therefore, say, in general, that softening of the heart is a very dangerous affection, and the more so, as it may cause death by rupture.

The *treatment* of softening is only that of the diseases which have caused it: as to idiopathic softening, its treatment corresponds to that of dilatation.

CHAPTER VI.

INDURATION OF THE HEART.

The muscular substance of the heart is susceptible of induration. This change, which most commonly coincides with hypertrophy, is of rare occurrence: generally it occupies the left ventricle only, and of it but a very small portion. It may, however, be general: Corvisart has seen the whole heart become indurated, so as to become resonant on percussion; the scalpel in cutting it met with great resistance, and gave rise to, perceptibly, a peculiar crepitous sound; still, the fleshy substance of the heart had its natural colour, and did not seem to have undergone any kind of transformation. Induration is not, as Laennec thinks, the result of an increase in nutrition, but rather a perversion of nutrition, which is most commonly connected with chronic inflammation of the heart. This author referred to induration of this organ a considerable augmentation of its impulse in this point; but we may, perhaps, be permitted to believe that Laennec was influenced too much

by speculative views. The impulse bears a relation to the rapidity and extent of the contractions of the heart; in induration, the contraction becomes difficult, and the movements are limited. It is then probable that the impulse would lose its energy.

The treatment of this affection is identical with that of hypertrophy.

CHAPTER VII.

EXCESSIVE DEVELOPMENT AND FATTY DEGENERATIONS OF THE HEART.

In persons of a certain age, who are very fat, and, in some exceptional circumstances, in individuals who are only moderately corpulent, we find the heart considerably loaded with fat. It is of a fine straw-yellow colour, and of a good consistence, is generally accumulated at the base and posterior part of the heart, around the cardiac arteries, and in the depression which separates the auricles from the ventricles; it is sometimes in so large a quantity that the heart appears lost in the fatty mass which envelopes it. Generally the fat penetrates more or less deeply between the fibres of the heart, and the walls of this organ become attenuated and flaccid.

The old authors regarded this condition as the cause of more or less serious accidents, even of sudden death; such was also the opinion of Corvisart, but it has not been adopted by Laennec. At any rate, Hope thinks that the excess of fat may embarrass the motions of the heart and the act of circulation, and that it can be, as he has often, recognised it, by the following signs: 1st, feebleness of the sounds of the heart, especially of the first sound; 2d, irregularity of the pulse without valvular disease; 3d, feeling of oppression or uneasiness in the cardiac region; 4th, the impulse of the heart retaining its normal character. All these signs, added to the symptoms which are habitually referred to disorder of the circulation, are sufficient to admit the existence of an excess of fat in the heart, especially in large and robust persons. Theory appears to show that the replacement of the muscular tissue of the heart by adipose tissue, and the attenuation of the walls, which is often its consequence, would favour the rupture of this organ; but experience has not confirmed this idea, and although rupture has been found in the case of fatty hypertrophy, it has not been observed oftener, for example, than in the case of dilatation.

Fatty degeneration, much more rare than fatty hypertrophy, consists in an infiltration of the muscular texture of the heart by a substance which has the chemical and physical properties of fat. This alteration differs essentially from fatty hypertrophy, because, in

this last, the muscular fibres become atrophied without losing their proper characters, whilst, in the first affection, the muscular fibres become pale and acquire a yellow colour, analogous to that of dead leaves, and very similar to that of certain softened hearts. Fatty degeneration is ordinarily partial, and only occupies a very circumscribed portion of the ventricles; the apex, for example; although it has been seen to occupy the greatest part of the two ventricles. It is an alteration very similar to the fatty degeneration which Haller and Vicq d'Azyr have noticed in the muscles. Like this, it is distinguished from softening properly so called, because it greases very much the scalpel and blotting paper. We possess no sign, by which we can recognise this last alteration of the heart.

CHAPTER VIII.

OSSEOUS, CARTILAGINOUS, AND OTHER ACCIDENTAL PRODUCTIONS IN THE MUSCULAR TISSUE OF THE HEART AND PERICARDIUM.

Osseous or cartilaginous productions are seldom developed in the muscular substance itself; it is oftener in the cellular tissue subjacent to the external or internal membrane of the heart that they are first deposited, whence they send prolongations between the muscular fasciculi. A part of the ventricle or auricle has been seen, in which the *columnæ carneæ* were transformed into cartilage, or completely ossified. M. Renauldin has seen the left ventricle transferred into a true petrification, which had a sabulous appearance in certain places, and, in others, resembled a saline crystallisation; and Burns has found the ventricles completely ossified in their whole extent, and resembling perfectly the bones of the cranium. These transformations are generally the result of a pericarditis or endocarditis, and principally affect the left ventricle. Under certain circumstances we find them in old men; and it is very difficult to refer them to anything more than a perversion of the normal nutrition.

Osseous or cartilaginous formations in the muscular substance, all tend, as Corvisart thought, to embarrass the movements and contractions of the heart, but not to increase the strength of the pulsations, and the intensity of the sounds, as was supposed by Laennec. The sounds and the impulse in this case are evidently enfeebled. The general and local symptoms which characterise these alterations, have been too imperfectly studied for us to be able to speak of them with any degree of certainty. It is probable, however, that the cartilaginous and osseous transformations of the muscular substance end by giving rise to murmurs, because these alterations are not long in extending to the valves; and,

in the contrary case, where the alterations are developed, especially in the pericardium, the friction sound should be heard. We sometimes find osseous or cartilaginous productions in the reflected fold of the pericardium, although most generally they have their original seat in the sub-serous cellular tissue, or in the fibrous membrane. These accidental ossifications of the pericardium, which are always very rare, vary much in extent and thickness; in some cases they send prolongations even into the muscular substance of the heart. They ordinarily occasion all those general symptoms which indicate great disorder in the circulation, and a friction sound which is always rough. Dr. Elliotson has reported two cases, in which masses of cartilage deposited in the pericardium have compressed the pulmonary artery, and caused the production of a murmur.

Cancer of the Heart and Pericardium. — These are very rare affections. The two common forms of cancer, scirrhus and encephaloid, have been found in the heart. Encephaloid cancer, which is the least rare, forms small masses in the muscular substance of the ventricles, or rather it is deposited in layers around the cardiac vessels, and covered by the pericardium, which is itself more or less altered. The cancerous matter commonly produces at the surface of the hearts swellings whose size is considerable; sometimes the fleshy fibres of this organ are atrophied or destroyed; sometimes they are hypertrophied. This degeneration of the heart is seldom seen without similar alterations in the neighbouring organs, especially in the lungs. We possess no sign whereby to recognise this disease.

Tubercles, Hydatids, Cysts, Melanosis of the Heart and the Pericardium.—We find, sometimes, tubercles disseminated through the thickness of the muscular substance. These cases are rare, and with them, most frequently, there are tubercles in the cellular tissue subjacent to the pericardium, in this membrane itself, in the false membranes which it encloses, and finally, in the lungs.

Hydatids of the species *cysticeric* have been noticed in the thickness of the walls of the heart. Finally, authors speak of serous cysts which have their seat between the muscular substance and the serous pericardium, and which in some circumstances are inserted into, and project from, the internal surface of the heart. There have also been found in the cavity of the pericardium serous cysts whose origin is entirely unknown. M. Cruveilhier has mentioned one case of melanosis of the heart and pericardium.

CHAPTER IX.

DISEASES OF THE VALVES AND ORIFICES OF THE HEART.

Anatomical Characters. — It is well known that the valves and the *chordæ tendineæ* of the heart are composed of a lamina of fibrous tissue, covered by the internal membrane of the heart. The fibrous tissue is the prolongation of a whitish and dense zone of the same nature, which surrounds each orifice of the heart, and which serves as a point of insertion to the muscular fibres of this organ. The internal membrane of the heart resembles the serous membranes. We may, therefore, consider the valves as formed of *fibro-serous tissues.* Now the fibrous tissue is remarkable for the facility with which it passes into the cartilaginous and osseous state; and this explains to us why the valves and orifices of the heart are often thus affected; and why the cavities which are only covered by the internal membrane are so rarely attacked. A valve and tendinous chord may be altered in their whole extent; but the alteration stops suddenly at the point where the serous membrane is detached from the circular zone, and where the *chordæ tendineæ* come out from the muscular substance. Sometimes, however, the alteration exceeds these limits; but the internal membrane which lines the muscular substance is never affected, unless the disease be originally developed in the valves.

It is, therefore, in the fibrous and not in the serous tissue that the disease commences. It is also in those points where the fibrous tissue is most abundant (that is, the base and free border of the valves) that the cartilaginous and osseous degenerations are most frequent and extensive. The serous membrane very often preserves its transparency.

The diseases of the valves are much more common in the left than in the right side of the heart; being as sixteen to one, according to Dr. Clendinning, and twenty to one, according to Hope. It may be remarked, that we seldom see either fibrous or cartilaginous, or osseous indurations, in the right side of the heart; and when both sides are affected at once, it seldom happens that the disease of the right is more advanced than the disease of the left side. Authors do not agree as to the cause of this difference. Some (Corvisart and Hope) attribute it to the more decidedly fibrous organization of the valves of the left side of the heart, by virtue of which they are more disposed to receive, either the materials which would transform them into cartilage, or calcareous salts destined to give them an osseous or stony hardness. Other authors (MM. Bertin and Bouillaud) ascribe it to the more vital, more stimulating and irritating qualities of the blood, which traverses the left cavities of the heart.

When the diseases of the valves are the result of chronic endocarditis, which is most often the case, we find traces of old inflammation of the serous membrane. The internal membrane presents more or less deep redness, an increased thickness, with loss of its transparency and habitual polish, more or less extensive false membranes, resembling milky spots on the pericardium, adhesions of the valves to each other and to the walls of the heart, &c. There is no doubt that, in a certain number of cases, the inflamed membrane and its secretory products do undergo analogous transformations; that is, pass into the fibrous, fibro-cartilaginous, or osseous state; but we have no proof that this transformation takes place under the influence of inflammation; and, besides, as diseases of the valves supervene sometimes without any appreciable phlegmasia, we have thought proper to separate these disorders from chronic endocarditis.

Alterations of the valves and of the orifices of the heart affect in various ways. They oppose the passage of the blood (*contraction*); or they prevent the play of the valves, and permit the reflux of blood into the cavities which it has just left (*regurgitation*): finally, they may both contract the orifice and impede the action of the valves (*contraction with regurgitation*). This last alteration is the most frequent. Simple contractions are much more uncommon than regurgitation; and this is easily understood, since a very slight alteration is sufficient to cause regurgitation, whilst contractions are always the result of a prolonged organic lesion.

Alterations of the valves, considered in a general manner, are not all equally frequent: we have already said, that the diseases of the valves of the heart were rare. Numerous researches have taught us that the mitral valve is oftener affected in the adult than the other valves, and that, after the mitral valve, the aortic, tricuspid, and pulmonary valves are next affected. In old persons, the diseases of the aortic valves are almost as common as diseases of the mitral.

Their anatomical alterations correspond with the differences which naturally exist between the auriculo-ventricular valves and the arterial valves. This induces us to describe separately their morbid changes.

1st. *Diseases of the Mitral Valve.* — The structural lesions vary, according as they occupy the base, the free border, or the whole of this valve. When the mitral valve has undergone the fibro-cartilaginous degeneration in its whole extent, it becomes contracted on itself, and what it loses in extent, it gains, so to speak, in the thickness of its free border; this last has sometimes the form of a ring or of an oval; at other times, that of a transverse slit, like a button-hole. The diameter of the aperture varies from a quarter of an inch to an inch. The thickness of the border varies equally. From this contraction of the valve there results a kind of funnel, which projects into the cavity of the ventricle, and sometimes so far that the *columnæ carneæ* are almost immediately inserted

into the ring, the *chordæ tendineæ* having disappeared. So long as the induration of the valve is not accompanied by excrescences, and especially ossiform ones, its surface is smooth, polished, and transparent. The presence of calcareous products, and of more or less prominent excrescences, before long induces the absorption of the internal membrane; and hence they come in contact with the blood. The ossifications which are produced under these circumstances present some varieties; they are either developed in a cartilaginous mass, or the calcareous matters are deposited at once in the subserous cellular tissue, in the for ofm polished and semi-transparent scales, or of small, opaque, yellowish granulations.

Sometimes all the membranous portion, and the free border of a valve are healthy, while the fibrous zone of the base has passed into a cartilaginous state, or it is covered with small calcareous incrustations, or converted, throughout its whole thickness, in a very thick, osseous ring. The deposits which take place at the base of the valve contract more or less the orifice, at the same time that the valve loses its freedom of movement.

At other times the base and the middle of the valve are sound, and the free border alone is diseased: very often the free extremity is covered with vegetations, with little cartilaginous nodules, with rounded calcareous granules, which prevent the two segments of the valve from approaching each other, and consequently produce regurgitation. Sometimes the only alteration of this valve consists in the hardening and thickening of the *chordæ tendineæ*, which prevents complete closure; at other times the regurgitation results from one of the segments of the valve having contracted adhesions with the walls of the ventricles. It is commonly the posterior segment, because it is the least moveable. Another affection of the mitral valve, which may cause regurgitation, which is best seen towards the semilunar valves of the aorta, consists in the atrophy of its membranous folds: then the valves may be reduced to a true reticulated tissue, often even presenting numerous perforations.

Finally, every time that an orifice of the heart becomes evidently dilated, without a corresponding dilatation of the valves, there is regurgitation; this holds true as well in regard to the mitral valve as any other.

Diseases of the Valves of the Aorta. — The indurations of the semilunar valves of the aorta, like those of the mitral valve, are more frequent and more extensive at the base and free border than in the median part of its membranous folds. When they occupy the free border, they are commonly developed in the sesamoid tubercles of these valves, which are often converted into a fibro-cartilaginous ring, resulting from the soldering of the three segments of this valve. At other times, the extremity of each one of these valves in particular is thickened, and contracted so as to leave, in the centre, a certain space which permits regurgitation.

The valves are frequently thickened and deformed in consequence

of the deposit of osteo-cartilaginous matter which gives them a yellowish, opaque colour. Succeeding to these degenerations, the angles of the valves become detached from their base, so that, only now holding by their centre, they have no fixed support in order to resist the reflux of the blood. It may even happen that the basis of all the valves are detached through almost their whole extent.

Sometimes, also, consequent on inflammation, the valves contract adhesions with the walls of the arteries, whence results an interval between them which permits regurgitation. This may also be produced by the rupture of the border of a valve.

Such are the cartilaginous and steatomatous degenerations which affect the valves of the aorta. As to ossifications, they are much more frequent in the valves of the aorta than in the mitral valve. They are rarely uniform in their appearance or in their composition; they often contain a great proportion of cartilaginous matter. These are ordinarily concretions of variable size, which arise either from the extremity of the valves, or from their base; in some cases the fibrous zone which surrounds the base of the orifice of the aorta may be converted into a very thick osseous ring.

When the ossifications are limited to the free extremity and to the base, whilst the middle part remains sound to a certain extent, the valve may still fulfil its functions, and only slightly impede the circulation; but when the middle part of the valves is ossified, they unite together, or they are curved upon themselves in the direction of their convexity or concavity, so as to represent certain sea shells.

In these different situations they may become immoveable. If curled forwards, they are applied to the walls of the aorta, and present no other obstacle to the passage of the blood than that which is produced by their size. Thence results regurgitation. If curling in the opposite direction, they remain fixed and immoveable in the closed position, and contract considerably the orifice, at the same time that they allow regurgitation. It is not uncommon to see a valve curled in an opposite direction from the other two. All three valves have been seen ossified in the closed position; one alone preserved slight mobility at its base for the passage of the blood by a narrow aperture.

It is in the semilunar valves, that an alteration, described under the name of *atrophy*, or *reticular condition*, is best seen, which consists in perforations or little apertures, almost always occupying the middle of these valves. The apertures, which sometimes are only the diameter of a needle-hole, and which are then so numerous as to perforate these membranous folds with a number of small holes, may be at other times single, and sufficiently large to occupy all the space included between the adherent and free border of the valves. In this last case the aperture, which is oval, is traversed by small filaments arising from the corpusculum Aurantii, and which are parallel to the free extremity of the valve affected.

Independently of the alterations which belong to the mitral valve

and to the valves of the aorta, there exist some alterations common to both these valves; we allude to *vegetations*, which have been divided into *warty* and into *globular*. The *first* have a very marked resemblance to venereal warts; they exhibit an irregularly spherical, ovoid or cylindrical form; their size varies from that of a pin's head to that of a large pea, and even of a horse bean; their surface is polished, but often lobulated like that of a mulberry; they are isolated or in groups; or in closely agglomerated patches like cauliflowers. Sometimes single, they are at others so numerous as to cover all the valves, the *chordæ tendineæ*, and a large portion of the auricle. Their colour, which is ordinarily grayish or yellowish, often acquires, to a greater or less extent, a pink, or more or less deep red colour. They are soft and slightly transparent. In general they are humid, as if their organization was not complete, and they may be easily detached with the handle of the scalpel; at other times they are firm like fibro-cartilage, creak under the scalpel, and can with difficulty be detached. The more firm in texture the vegetations, the larger and more warty they are. Almost always the internal membrane of the heart is diseased in the part from which the vegetations spring; it is thickened, steatomatous, or cartilaginous; sometimes ossified, ulcerated, or eroded. When the internal membrane is destroyed in one part, the vegetations may be numerous, or limited to the diseased part, and much more voluminous than common. It is especially at the base and free extremity of the valves that the warty vegetations are developed. In these points, especially in the last, they are often disposed in rings; more frequently occupying the left than the right side of the heart, and the valves than the auricles. When they have their seat at the base or free border of a valve, they impede valvular movement, prevent closure, and contract more or less the diameter of the orifice, in proportion to their size and number. What is the nature of these vegetations? Laennec regarded them as small polypous or fibrinous concretions, which become organised by a process of absorption or nutrition, analogous to that which converts albuminous false membranes into adventitious membranes or into cellular tissue. But, if we reflect that polypous concretions are most frequently present in the right cavities, whilst the warty vegetations there are rare; if we consider that the essential condition of the coagulation of the blood, and of the formation of concretions is stagnation; whereas, it is in the valves (the most moveable parts of the heart) that these vegetations are ordinarily developed, we cannot hesitate to regard this conclusion as erroneous. Kreysig, MM. Bertin and Bouillaud have referred the development of these vegetations to inflammation. Corvisart regarded them as of syphilitic origin.

The *second* (*globular vegetations*) are presented in the form of small globes or cysts, irregularly spherical or ovoidal, whose size varies from that of a pea to that of a pigeon's egg. Smooth on the exterior, these small cysts, having less than a line in dia-

meter, are organised, a little firmer than the white of an egg, and resemble, from their opacity, the oldest polypous concretions. The internal surface of the cysts, slightly smoother than the external, is formed of a softer substance, which, in proportion as we approach the centre of the tumour, is found to consist of a matter similar to that which is enclosed in its interor. This is sometimes blood, sometimes a substance analogous to the *lees* of wine, and sometimes pus. These cysts adhere to the *columnæ carneæ*, or to the internal membrane of the heart, by a pedicle of a more recent formation, more transparent, and of a less advanced organisation than the cyst itself. We never meet with them except in the ventricles, and the sinuses of the auricles. We find them as often in the right cavities as in the left; but it is especially at the inferior part of the ventricles, and quite near the apex, that these small bodies are commonly placed. It does not appear that they have been observed in the large vessels. As to their nature, Laennec does not clearly speak; they are now generally regarded as polypous concretions which have suppurated in their centre through the medium of an inflammatory process.

Diseases of the Valves of the Right Side of the Heart. — These diseases, as we have already said, are less frequently met with than those of the left side of the heart, and most generally are less advanced in their development. They nearly always exist in connection with the diseases of the valves of the left side of the heart. The tricuspid valve is oftener altered than the valves of the pulmonary artery, at least after birth, and the cartilaginous is oftener observed than the osseous induration. The alterations of the pulmonary artery are rarely observed in adults. They are at their maximum in individuals who present congenital communications between the different cavities of the heart. In other respects, the diseases of the valves of the right heart, whatever be their nature, only differ from those of the left side in their frequency and extent; their characters are essentially the same.

Causes. — 1st. *Predisposing:* the more fibrous structure of the valves of the left side of the heart, advanced age, and all cachexies, whatever be their origin. 2d. *Determining:* endocarditis, and especially rheumatic endocarditis. All the causes which increase the momentum of the circulation, and which diminish the play of the valves; such as exercise of the body when violent and repeated, hypertrophy with dilatation, nervous and prolonged palpitations, &c.

*Symptoms.** — A. *General Symptoms.* — As these symptoms do

* Every time that a valve is diseased, and the alteration of which it is the seat causes a contraction of the corresponding orifice, or embarrasses the play of the valves, so as to produce a regurgitation, or these two at once, there results a mechanical obstacle to the circulation, whence are derived in part the symptoms of the diseases of the valves. It is an error (as Hope has remarked), in MM. Bertin and Bouillaud, and M. Bouillaud in particular, to place the cause of the obstructed circulation in the alterations of the valves. It must also be found in the co-existing disease of the muscular apparatus. This is

not bear a relation to the nature of the alteration of the valves, but rather to the degree of contraction or of regurgitation, we shall divide them into two kinds: — 1st, general symptoms, common to these diseases and to dilatation of the heart: and 2d, general symptoms characteristic of these diseases.

1st. *Common general symptoms.* — These are cough, expectoration, dyspnœa, orthopnœa, frighful dreams, sudden starts in waking, œdema, pulmonary congestion, and apoplexy, passive hæmoptysis, swelling of the jugulars, lividity of the face, anasarca, and other forms of dropsy, injection of many or all the mucous membranes, engorgement of the spleen, &c., cerebral congestion, sometimes even apoplexy. All these symptoms are never seen united except in very severe cases, and at a very advanced stage of the disease.

2d. *Characteristic general symptoms.* — These are: *a*, the *intensity* of all the preceding symptoms, in particular of dyspnœa, especially when hypertrophy or dilatation is joined to the disease of the valves, an intensity which is much greater than in the case of hypertrophy or simple dilatation in the same degree; *b*, *colliquative diuresis*, which is principally seen in diseases of the valves, and especially in the diseases of either of the auriculo-ventricular orifices (Gendrin); *c*, *swelling of the jugular veins with pulsations synchronous* with the systole of the arteries, which most often depends on regurgitation through the tricuspid valve; *d*, *the state of the pulse.* There is perhaps no point on which authors agree less than on the value of an examination of the pulse, in the diagnosis of diseases of the valves. Some attach importance to this examination; others seek to prove that it is without avail; others, finally, contend that in all the diseases of the valves

a fact, which we have very often verified; the diseases of the valves are very supportable, when there is neither dilatation, nor hypertrophy of the cavities of the heart, nor softening of the muscular substance; but as soon as these complications supervene, the symptoms of disordered circulation are developed, and early attain their maximum of intensity. It cannot then be doubted that the grave symptoms which are observed in the diseases of the valves are to be attributed in a great measure to hypertrophy or to dilatation. It is, however, supposable that a contraction extending very far, might, of *itself*, induce all these symptoms. But as an extreme contraction never exists without an alteration in the muscular walls of the heart, it is impossible to justify this hypothesis. The serious character which the alterations of the muscular substance of the heart impress on the disease of the valves, is a fact that is very important to be known, since it teaches us that we should use all our exertions to guard against these complications. It remains to know how dilatation and hypertrophy aggravate the symptoms of the diseases of the valves. "Dilatation," says Hope, "by enfeebling the contractility of the heart, constitutes an obstacle to the circulation as well as any mechanical obstacle; it is clear that the symptoms, having a double origin, should be doubly grave. In hypertrophy, the heart, endowed with a morbid contractility, contends against the obstacle, and there is presented in too great quantity to the contracted orifice, or results palpitations. Now, as while they last, the blood flows back in too great abundance through the imperfect orifice, the circulation is performed with a difficulty which is always increasing."

there is one and the same state of the pulse. The numerous researches which we have made, for three years past, on the pathology of the heart, lead us to the same results with Dr. Hope, and justify us in deducing the following conclusions:—1. The irregularity with intermission, inequality and feebleness of the pulse which is met with in the diseases of the heart, is principally seen in the contraction or regurgitation of the left auriculo-ventricular orifice or mitral valve. These characters of the pulse are also met with in contraction of the aortic orifice, but only when this contraction is *extreme*. 2. Regurgitation through the valves of the aorta impresses on the pulse a very bounding character without irregularities; that is, the arterial diastole is short and quick, as if the blood was thrown out suddenly and by jerks. At a more advanced stage of the disease, when the arterial tubes become hypertrophied, the arterial diastole is prolonged, so that the artery no longer collapses after each diastole, which seems now to be but a simple increase of the preceding diastole. 3. No importance should be attached to the modifications in force, in extent and rhythm, of the pulse during the attacks of palpitation and dyspnœa, and in the last stages of the diseases of the heart. 4. The diseases of the valves of the right side of the heart exert scarcely any effect on the state of the pulse.

B. *Local Symptoms.* — 1st. *Physiological Symptoms.* The *pain* which the patients feel in the precordial region is variable in its intensity; sometimes it is lancinating, and acquires extreme sharpness during the attacks of palpitations. The *palpitations* which are met with in the diseases of the valves are reproduced with the greatest facility, and are of extreme tenacity: they ordinarily appear in paroxysms, especially at night, or after slightly violent exertions. The *impulse* of the heart undergoes no other modifications than those which result from the complications that ordinarily accompany diseases of the valves.

2d. *Physical Symptoms.— Percussion.* The dulness of the precordial region is always in relation with the size of the central organ of the circulation: it increases in the diseases of the valves, in consequence of the increase of size which the heart acquires.

Auscultation. — We have studied the nature, causes, and mechanism of the bellows, filing, sawing, and rasping sounds, and the musical or sibilant murmurs, which are produced in the diseases of the valves (*intra-cardiac murmurs from organic cause*): we have indicated the points at which they should be sought: we have also studied the vibratory tremor. It remains for us to show in what manner these phenomena are associated to constitute the signs of the diseases of the valves in particular.

Physical symptoms of the diseases of the valves of the aorta: 1. In *contraction of the aortic orifice.* There is heard during the contraction of the ventricles, that is to say, with the first sound, near the sternum, on a level with the inferior border of the third rib, and thence ascending to the extent of two inches along the course of

the aorta, a murmur whose nature varies, and whose maximum is found on a level with the points which we have indicated.

This *systolic murmur* is distinguished from the one which belongs to diseases of the aorta itself, by this last having its maximum rather along the course of the ascending aorta than opposite to the valves; and by its being, at the same time, much more superficial and much nearer. In some cases, especially when the heart is displaced from beneath the sternum (Hope), there is perceived a *vibratory* or *purring tremor* coinciding with the systole. 2. In *regurgitation through the valves of the aorta*, the second sound of the heart is accompanied by a murmur which presents the following characters: *a*, it is louder and more superficial on a level with and above the valves of the aorta than towards the apex of the heart, which distinguishes it essentially from the diastolic murmurs that belong to the left auriculo-ventricular orifice: *b*, it is louder along the course of the ascending aorta than that of the pulmonary artery, along the course of the left ventricle than that of the right, which prevents its being confounded with the murmurs that belong to the valves of the pulmonary artery: *c*, it is distinguished from a systolic murmur in the aortic orifice, by its coinciding with the second sound; in being perceived a little lower on the course of the ventricle than the systolic murmurs; by its being prolonged during the whole interval of repose, and even during the accidental intermissions of the ventricular contraction; by ordinarily having a softer character and lower tone; and finally, by often acquiring the musical character. The diastolic murmur is prolonged in the aorta, as we have already said; but it would soon cease if it was not replaced by a *blowing sound*, which is produced in the ascending aorta, the carotids and subclavian, and which coincides with the arterial diastole, and systole of the ventricles. The existence of this diastolic bellows sounds in the arteries of the neck, in an individual who does not present any trace of cachexia, or of disease of the arteries, is a sign of great importance in the diagnosis of regurgitation through the valves of the aorta. The *vibratory tremor* is never perceived in simple regurgitation. 3. In *contraction with regurgitation through the aortic orifice*, there is heard, on a level with the inferior border of the third rib, near the sternum, a *double systolic and diastolic murmur*, which constitutes a sound of two tones connected with each other so as to form an advancing and receding sound (Gendrin), and which is prolonged for the extent of two to three inches along the course of the aorta. There is also heard, under some circumstances, a *systolic vibratory tremor;* finally, we find in the arteries a blowing murmur coinciding with the arterial diastole.

Physical symptoms of the diseases of the mitral valve: 1st. When the *auriculo-ventricular orifice is much contracted*, the ventricular diastole and second sound are accompanied by a *murmur*, whose maximum intensity is found at the apex of the heart: it is distinguishable by its seat from the diastolic murmurs that belong

to the orifice of the aorta. This murmur extends outwards and to the left of the heart, and is thus prevented from being confounded with the murmurs of the right auriculo-ventricular orifice; it is always very feeble and soft, like the bellows sound, owing to the little force with which the diastolic current is endowed. This murmur is often wanting, when the contraction is slightly marked, and in some circumstances where the contraction is considerable, it may also be wanting; for example, when the heart is much enfeebled by dilatation or softening. We never find the vibratory tremor in contraction of the left auriculo-ventricular orifice. 2. *In regurgitation through the mitral valve*, the first sound is accompanied by a *murmur* whose nature varies in relation to the obstacle: its tone is very low, and it is at its maximum on a level with the apex of the heart, and a little without and to the left of the mamma. This murmur is distinguished by its situation from the systolic murmur which belong to the aortic orifice. Finally, *a systolic vibratory tremor* is often heard, especially when the left ventricle is affected with hypertrophy and dilatation. 3. *In contraction with regurgitation through the left auriculo-ventricular orifice*, there may be found a *double diastolic and systolic murmur*, having its maximum at the apex of the heart; but more commonly there is only a *systolic murmur*, indicating regurgitation, always in consequence of the feebleness of the current which comes from the auricle. This murmur is frequently accompanied by a *systolic vibratory tremor.*

Physical symptoms of the diseases of the valves of the pulmonary artery: 1. When the *pulmonary orifice is contracted*, the signs are the same as in contraction of the aortic orifice; that is to say, that the first sound is accompanied by a *murmur*, with this difference, that the pulmonary artery being more superficial, the murmur is nearer the ear, and of a more elevated pitch, than that of the aortic orifice. This murmur, corresponding with the first sound, is distinguished from that which takes place in the aorta, by its being much louder along the course of the pulmonary artery than on that of the aorta, and upon the course of the right ventricle than on that of the left ventricle. It is also distinguished from that which indicates regurgitation through the auriculo-ventricular orifice, because it is perceived on the course of the pulmonary artery at a point where the murmur belonging to the mitral valve cannot be heard. When this systolic murmur or purring tremor of the artery has its maximum between the second and third ribs, close to the sternum, and not on a level with the inferior border of the third rib opposite to the valves, we may suspect a dilatation of the pulmonary artery. 2. *When the valves of the pulmonary artery are imperfect*, the second sound is accompanied by a *murmur* whose nature and diagnosis are absolutely the same as for the aorta, with this difference, that the diastolic blowing murmur of which we have spoken is not found in the arteries, nor the bounding pulse, which are characteristic of regurgitation through the aorta. The diseases of the pulmonary artery are so rare, that their existence ought never to be affirmed, unless

the signs which we have just enumerated are found to be completely and clearly marked; or unless the signs of a communication between the sides of the heart are evident; for, in these last circumstances, the pulmonary orifice is commonly contracted.

Physical symptoms of the diseases of the tricuspid valve. — These signs are the same as for the diseases of the mitral valve, except that the murmurs to which they give rise are best heard along the course of or near to the sternum, on the same level as in the case of disease of the mitral valve, that is, on a level with or a little above the apex of the heart. The pulse never presents as much irregularity, nor has the purring tremor ever been observed in these cases. The tricuspid valve is rarely so far diseased as to occasion a murmur: its lesions are not common, with the exception, perhaps, of regurgitation; and therefore the practitioner should be very careful in his diagnosis of the diseases of this valve.

To recapitulate, we find that:

- **The murmurs corresponding with the first or systolic sound, indicate**
  - **contraction of the arterial orifices.**
    - **aortic.** — Maximum of the murmur on a level with the inferior edge of the third rib, and thence ascending to the right along the course of the aorta.
    - **pulmonary.** — Maximum of the murmur along the course of the pulmonary artery; murmur of a more elevated pitch and nearer the ear than the systolic murmur of the aortic orifice.
  - **regurgitation through the valves**
    - **mitral.** — Maximum of the murmur on a level with or a little above the apex of the heart, extending outwards and to the left. This murmur is deep and of a more elevated pitch; it is often accompanied by the vibratory or purring tremor.
    - **tricuspid.** — Maximum of the murmur along the course of, or near the left border of the sternum; on a level with or a little above the apex of the heart.
- **The murmurs corresponding to the second or diastolic sound, indicate**
  - **deficiency of the valves.**
    - **aortic.** — Maximum of the murmur on a level with the inferior border of the third rib, and a little above this point, along the course of the aorta. This murmur is better perceived along the left ventricle, than the systolic murmur of the same orifice.
    - **pulmonary.** — Maximum of the murmur on a level with the inferior border of the third rib, along the course of the pulmonary artery, slightly along that of the right ventricle.
  - **contraction of the orifices.**
    - **left auriculo-ventricular.** — Maximum of the murmur on a level with or a little above the apex of the heart, extending outwards and to the left; always feeble and low, it is wanting when the contraction is not very considerable.
    - **right auriculo-ventricular** — Maximum of the murmur on a level with or a little above the apex of the heart, but nearer the sternum. Like the preceding it is often wanting.

Physical symptoms of the diseases of the arterial and auriculo-ventricular valves occurring together.—The murmurs which we have described separately for each one of these diseases, are found united, and the whole nicety of the diagnosis consists in determining the maximum degree of these murmurs. This will be easy, as all the murmurs do not present the same character of roughness or softness.

Diagnosis. — In order to form a sure diagnosis in the diseases of the valves, we must ascertain, 1st, that a disease of the valves exists, which is easily recognised by the intimate nature of the murmur; 2d, which is the orifice or cavity affected, to be determined by the maximum degree of the murmur; 3d, whether there be regurgitation or contraction, which is ascertained by the synchronism of the murmur with one of the two sounds of the heart; 4th, what is the nature of the alteration (cartilaginous or osseous induration), to be ascertained by the tone of the murmur; 5th, whether there exists any complication, (hypertrophy, dilatation, diseases of the aorta,) to be characterized by its proper signs.

Progress, duration, terminations. — The *progress* of the diseases of the valves is ordinarily continuous, and their course is always rapid. It is not, however, rare to see, at the commencement, these diseases only announced by intermittent symptoms, whatever may be the gravity of the organic lesion; but after a certain time they resume their continued march, and all the severer symptoms of which we have spoken are presented in succession. Diseases of the valves are rarely limited to the valve which is primitively affected. Under some circumstances, the disease extends, as it were by continuity, to the neighbouring valve. It is thus that alterations of the mitral valve, and in particular of its anterior segment, are seen to extend to the valves of the aorta, and reciprocally. The valves may also be affected secondarily, in consequence of obstacles to the accomplishment of their functions. It is thus, that, when the left auriculo-ventricular orifice is diseased, the tricuspid valve almost always becomes inefficient, in consequence of the obstruction to its movements, from atrophy of its *columnæ carneæ*, and in consequence of its membranous segments. The *duration* of the diseases of the valves is unlimited. In individuals who are able to procure repose, and willing to submit to all the exigencies of regimen and treatment, these diseases are often seen to last from ten to twenty years. It is impossible to say, in a positive manner, what will be the *termination* of a valvular disease. Termination by resolution is very rare, especially when the induration is fibro-cartilaginous or osseous; that by death is the most frequent. Death may be sudden; and this termination is principally observed in that form of disease which impedes so strongly the repletion of the arterial tubes, that is, regurgitation through the valves of the aorta. A recent example (the death of M. Humann, Minister of Finance) confirms the opinion, which we have heard professed for a long time by M. Gendrin. This unhappy termination may take place in the midst of exertion

or of violent exercise, at a time when the disease does not appear much advanced; because the embarrassment of the circulation has been suddenly carried to an extreme degree, and because the heart is no longer able to continue its functions. Apoplexy or cerebral congestion may cause a like result. Most commonly, however, in this last case, the patient remains for twelve or twenty-four hours, and sometimes for two or three days, in a comatose state, before he dies. Finally, death may take place in anemic patients from syncope. Termination by sudden death is not common; most generally the patient dies after having gone through all the stages of the disease, and his death is announced by the usual precursory phenomena. These rapid terminations are most frequently observed in persons accustomed to hard labour.

Prognosis. — The prognosis of diseases of the valves is generally unfavourable. It varies according to, 1st, the condition of the patient: young persons suffer much more than old persons from obstacles to the circulation. 2d. The nature of the alteration; contractions of the left auriculo-ventricular orifice and regurgitations through the valves of the aorta are the most dangerous. Alterations of an inflammatory nature kill sooner than those which are developed from an unknown cause. The osseous or cartilaginous indurations are beyond the resources of art. 3d. The complications which are observed in the heart or other organs; the diseases of the valves once developed are not long without causing hypertrophy of the cavity situated behind the obstacle. As long as this hypertrophy is slightly marked, it produces little or no inconvenience to the general health: but as soon as it becomes considerable, the disease runs rapidly to a fatal termination. This is also the case when either the cavities are attenuated or the walls become softened. The accidents which are naturally observed in the progress of the disease in the lungs or brain, add much to the gravity of the disease. Finally, in some cases, the serous membrane of the pericardium becomes inflamed without any known cause, *constituting* one of the most dangerous complications.

Treatment. — The first indication is to remove the disease which preceded that of the valves. When valvular disease succeeds to an acute endocarditis, when it is seen, as it were, to be developed under our eyes, it is necessary to continue the treatment employed in acute endocarditis, adapting it to the new conditions in which the patient is placed. This treatment should be continued, moreover, as long as we have reason to suppose that the inflammatory state persists.

When the diseases of the valves are developed under the influence of causes not inflammatory, they are most commonly not recognised till their maturity, and then it is beyond our pewer to destroy the cause which produced them. In the present state of the science we do not possess any means to remove a *confirmed* disease of the valves. We can only prevent its increase, oppose the tendency to complications (hypertrophy, dilatation, &c.), palliate the symptoms which result from obstructed circulation, and finally, to

retard indefinitely a fatal termination. Moderate bleedings, repeated at distant intervals, frugal and slightly stimulating repasts, but yet sufficiently invigorating quiet of body and mind; such is the basis of the treatment to be directed against diseases of the valves. In general, bleedings should be practised to diminish the hypertrophy of the walls of the heart, or the congestion which takes place in the interior of this organ; but it would be folly to employ sanguineous depletions according to the method of Albertini and of Valsalva, with the entirely chimerical design of obtaining the cure of the valvular alteration. In all diseases which enfeeble the contractile action of the heart (dilatation), and which diminish the quantity of blood that should normally penetrate into the arterial tubes (regurgitation through the valves of the aorta, for example), we should abstain as much as possible from sanguineous depletions, which might induce syncope or sudden death. The same rule should be observed with respect to old persons. Leeches or cups over the præcordial region may be added to general bleeding; and it will be found advantageous, especially when there is a chronic phlegmasia, to apply to the same part repeated blisters, which should be spread with powdered digitalis, or better still, a deep cautery. When there is dropsy, and the urine is in small quantity, diuretics are of great service; the acetate, nitrate and bitartrate of potassa, squills, and digitalis, are the means on which we can most rely. We should prescribe many at the same time, because diuretics are very variable in their effects; digitalis, which is one of the best, ought to be employed only for a certain period, and never when the contractility of the heart is much enfeebled; as in dilatation, for example. When the diuretics have not removed the dropsy, purgatives often succeed, principally the tincture of jalap, elaterium, &c.; the last in a dose of from an eighth to a fourth of a grain, and gradually to two grains. Diaphoretics may also be added to these measures, but only those which are neither active nor stimulating. When dropsy resists all these remedies, and the distension of the skin becomes extreme, punctures may be practised. The incision should never be made with the lancet or bistoury, because the escape of the liquids would take place very rapidly, and gangrene might attack the lips of the wound. It is sufficient to make, for some days, twenty to thirty punctures on the œdematous members with a canulated needle. Tonics — the different preparations of quinine, and of iron, are very useful in individuals enfeebled by dropsies, in whom the circulation is languid, and the whole system in a state of atony. Expectorants, such as tartar emetic, sulphuric ether, narcotics, (opium, hyosciamus, the smoke of the datura stramonium,) are proper in some cases of obstinate cardiac asthma. It must not be forgotten that these measures can only be auxiliary; that they act by diminishing the sensation of dyspnœa, but have no action on the essential cause of the dyspnœa. When, however, in the last stage of the disease, the sleeplessness and agitation are extreme, narcotics afford great relief, but only by producing sleep, and diminishing the suffering.

CHAPTER X.

ANEURISM OF THE AORTA.

Surgeons commonly describe, under the name of aneurism, all tumours formed by arterial blood, whether it be enclosed or not in the tubes which it naturally traverses. But this is not the meaning which we give to this word, and we shall only consider as aneurisms those tumours formed by the enlargement of a part or of the whole circumference of the vessel.

We will study successively — 1st, *aneurism from general dilatation*, that is, with enlargement of the whole circumference of an artery; 2d, *aneurism from partial dilatation*, or *true aneurism*; that is, the sacciform dilatation of a portion of the circumference, or of one side of the artery; 3d, *aneurism from rupture of the imternal and middle membrane*, or *false aneurism;* 4th, *mixed aneurism* or *false aneurism, formed on a true aneurism or on a dilatation.*

Anatomical Characters. — The names given to each one of these varietes alone suffice to make us understand the nature of the anatomical alterations peculiar to each of them.

General dilatation is characterised by a general increase in the diameter of the artery. Sometimes this latter presents in its interior some of the alterations which we have just described, as belonging to chronic arteritis (cartilaginous, steatomatous or calcareous deposits, thickening, contraction, fragility of the internal membrane, &c.). Sometimes, on the contrary, the membranes are in a healthy state. It is rare, however, that there is not found slight hypertrophy of the internal membrane and slight rigidity in the arterial tube. General dilatation may be uniform, and occupy a very large extent of the vessel, or merely consist of a swelling alone: it may also present several series of ovoidal or pyriform swellings. Although this dilatation does not generally exceed the natural diameter of the vessel, it may, however, extend far beyond it (three or four times its diameter). These dilatations seldom enclose laminated clots, unless there has been some ulceration, or some fissure of the internal membrane. In this last case, they are never so considerable as to obliterate completely the transparency of the vessel. General dilatation of the aorta has its principal seat in the ascending portion and its arch; sometimes it affects the descending or vertical portion of the thoracic or abdominal aorta. It commonly extends to the large trunks which spring from the dilated portion, with the exception perhaps of the left subclavian, which is almost always protected by its oblique insertion into the original trunk.

Partial dilatation, or *true aneurism*, differs from general dilatation by its occupying only a limited portion of the circumference of the

aorta, by terminating suddenly by a marked edge, and by having its neck more narrow than the fundus of the sac. It is distinguished from false aneurism, because the internal and middle membrane can be traced in its interior, and because it exhibits morbid productions which characterise the phlegmasiæ of these membranes. True aneurism varies in its size, from that of a walnut to that of the head of a fœtus at full term. Sometimes clots are found in true aneurisms; they are commonly in masses, very rarely lamellated, and adhere by limited points. These tumours most often occupy the ascending portion and the arch of the aorta, and are detached from the anterior or lateral portion of this vessel, whilst the posterior part and the concavity of the arch of the aorta are rarely affected by it. They are not observed as frequently as the preceding.

Every time that, from any cause, the internal and middle membranes undergo a solution of continuity, the blood, by its lateral impulse, raises the external membrane, and converts it into a sac which communicates with the interior of the artery by a contracted opening or neck; this is called *false aneurism* or *aneurism from rupture*.* Sometimes the external membrane, instead of widening in the form of a pouch, has been seen to be distended by degrees by the blood, to detach itself from the arterial walls to a very considerable extent. Finally, in one case, perhaps solitary, and which is due to Mr. Shekelton, the blood, after having followed this course to the extent of four inches, has been seen to penetrate anew through the internal and middle membrane, into the arterial canal, which was obliterated by the pressure of the tumour immediately above this opening. False aneurism presents no trace of an internal membrane. Its internal surface, lined with chronic inflammatory products, is covered by lameliform fibrinous clots, which contract adhesions with this surface. Its size is variable: it generally occupies the descending aorta, whilst aneurism from dilatation and true aneurism most generally are in the ascending portion and arch of the aorta.

If false be added to true aneurism, we have what is called *mixed aneurism;* that is to say, that the internal and middle coats of the dilated portion rupture, and the external membrane, alone supporting the lateral impulse, becomes dilated in the form of a superadded sac. These aneurisms, which are the most common, communicate with the interior of the aorta by a contracted opening, and present infinite varieties in their form, size, &c.

Considered in a general manner, the size of an aneurism is in a direct relation with the nature of the adjacent parts, or rather with the degree of extensibility with which they are endowed. In pro-

* Does there exist, as Haller, Dubois, and Dupuytren thought, a variety of aneurism in which the internal membrane makes a hernial projection through the torn fibrous membrane, and supports, with the cellular coat, the impulse of the blood? We do not think so; for the internal membrane of the arteries, as the experiments of Hunter, Scarpa, and Home, have proved, does not possess any elasticity, and will break sooner than be distended.

portion as the tumour progresses, the coats of the artery become attenuated and tear. The cellular coat, which alone remains, converts, by its dilatation, the true into a false aneurism. At a later period, this membrane breaks also, and the cellular sheath of the vessel alone opposes an obstacle to the effusion of blood. Finally, when this last yields in its turn, the contiguous parts, whatever may be their nature, contract adhesions with the tumour, and concur to form the parietes of the sac. The bones, and even the viscera themselves, may be included in the tumour. But when the adhesions are torn, the sac opens into the cavities of these organs — the lungs, œsophagus, intestines, stomach, bladder, &c., and most frequently causes immediate death. Among the alterations to which aneurisms give rise in adjacent parts, we will mention, as curious, erosions of the bones. Sometimes they are excavated, although still provided with their periosteum; sometimes the periosteum is destroyed, and they are deeply eroded, without any trace of suppuration, or any osseous destruction; which induces us to refer these alterations rather to a mechanical disaggregation, to a true wearing out, than to an inflammation, of which indeed there is no proof.

The clots which the aneurismal tumours enclose in their interior, differ according to their seat; the most concentric are formed by blood feebly coagulated. More outwardly, the clot is discoloured and drier; farther in, whitish or yellowish clots of pure fibrin are found; finally, in contact with the walls of the tumour, we discover clots of the same nature, but completely opaque, friable, and similar to pure fibrin. Feebly adherent to each other in their more concentric layers, the clots are more united outwardly by a fine wool, and like cellular tissue; and the adhesion is much more intimate in proportion to the duration of the clots. Generally speaking, the quantity and consistence of the clots are more considerable in proportion as the aperture which leads to the interior of the aneurism is smaller, and as the sac is riddled with a greater number of apertures.

Causes. — All the causes which increase the pressure of the blood on the aorta, or which diminish in some parts of its extent the resistance of this artery to the distending force of the blood, may cause an aneurism of it. Among the causes of the first kind, we place — hypertrophy of the left ventricle, obstacles to the arterial circulation, violent and prolonged efforts; among the second, all the diseases of the arterial walls which diminish their contractility.

Aneurisms of the aorta are more common among men than among women; but it does not appear that this difference is so great for aneurisms of the extremities.

Ruptures of aneurisms supervene under the influence of causes which increase the force of the arterial circulation (violent efforts, cries, &c.). This rupture may, however, be the result of the simple progress of the disease.

Symptoms. — A. *General Symptoms.* — The general symptoms are in a great measure identical with those of the organic diseases

of the heart. Palpitations, dyspnœa, cough, tendency to syncope, frightful dreams, sudden startings on awakening, hæmoptysis, livid colour of the face, cerebral and hepatic congestions, serous infiltration, &c., are observed. As might be inferred, the exciting cause of all these symptoms is always found in an obstacle to the circulation, depending on aneurism alone, or on aneurism with hypertrophy of the heart, to which latter aneurism gives rise sooner or later, especially if it occupies the ascending portion and the arch of the aorta. This complication of a disease of the muscular substance of the heart, gives greater intensity to the preceding symptoms; and explains to us how aneurismal tumours may last for years without producing much disorder, unless hypertrophy or dilatation be superadded. Among the general signs, some are more characteristic, especially when aided by stethoscopic signs. We may mention a *sensation of constriction and of fulness, irregularity of the pulse, slowness of the arterial diastole* at the wrist; whistling or rattling *respiration*, faint *voice, feebleness of the vesicular murmur* in one lung, and its *puerile character* in the other, extreme *dyspnœa*, often returning in paroxysms, *difficulty of deglutition*, severe *pain* in the spinal column, extending to the shoulder, neck, and arm, with a feeling of numbness, or pricking, and commencement of paralysis of this member; finally, a *varicose state* of the veins of either the superior or inferior portion of the trunk, resulting from the compression which the tumour makes on one of the large venous trunks.

B. *Local Symptoms.* — 1. *Physiological Symptoms.* — It is rare that true pain exists in the case of aneurism of the aorta. Most commonly there is merely a feeling of uneasiness and oppression in the part; although, under some circumstances the patients experience acute pain, in consequence of the compression which the tumour makes on certain organs. When the aneurism of the aorta is so large as to be in contact with the abdominal or thoracic parietes, there are perceived, in the place which the tremor occupies, simple beats, perfectly synchronous with the impulse of the heart, and often elevating the chest with great violence. In certain cases, there is observed a pulsatile *tumour* agitated by beats, synchronous with the ventricular systole. This last sign is one of those which furnish the strongest presumption in favour of the existence of an aneurism. Finally, there is sometimes seen, but only in aneurisms of the thoracic aorta, a true *arching* of the walls of the chest.

2. *Physical Signs.* — *Percussion.* — If the tumour be no larger than an egg, and so superficial as to be easily examined, although this be done by pressing on the parts external to it, an anomalous dulness is perceived. This dulness is rarely absolute, and bears a direct relation to the size of the tumour.

Auscultation. — When the ear or stethoscope is applied over an aneurismal tumour, a simple and sometimes double murmur is heard. Of these two murmurs, the first coincides with the systole of the ventricles, and the arterial diastole; the second, when it exists,

coincides with the arterial systole. The *diastolic murmur* of the aorta consists in a deep, harsh, very short sound, which commences and terminates suddenly, and which is sometimes more intense than the strongest cardiac murmurs. It sometimes resembles perfectly the sound which the passing of a file over a very hard body produces. This murmur is best observed in aneurism from dilatation, and it presents a rougher character according as the interior of the vessel is more studded with rough points, and especially osseous asperities. In old aneurisms, whose walls are thickened by fibrinous concretions, a dull and distant sound only is heard. The *systolic murmur* of the aneurism of the aorta, which depends either on the reaction of the arterial tissue against the blood that has just distended it, or on the reflux of the blood through a narrow aperture into the normal cavity of the vessel, never presents the rough character of the diastolic murmur: generally it has the character of the bellows sound; in some rare cases it is slightly sibilant. This systolic murmur is principally met with in considerable dilatation of the aorta and in the sacciform aneurism, but only when the polypous concretions are not numerous. It may disappear at a more or less advanced stage of the disease. This may also be the case with the diastolic murmur of sacciform aneurism, and when the aneurismal cavity is completely filled by clots. The *vibratory tremor* also constitutes a characteristic sign of the existence of an aneurism. It is much more marked in simple dilatation than in sacciform aneurisms, especially if the former is accompanied by numerous asperities on the internal surface of the artery. This murmur is often wanting in sacciform aneurism, because the greatest part of the blood silently continues its course along the smooth canal of the artery, and the blood enters the sac only in a small quantity. It is seldom observed in old aneurismal tumours. Such are the symptoms which belong to aneurism of the aorta, considered in a general manner. Let us now examine the local and general symptoms which belong to each one, in particular, of the varieties of aneurism of the aorta.

A. *Aneurism from dilatation of the arch and ascending portion of the aorta. — Local Symptoms.* — 1. Beats above the two clavicles, on a level with their sternal extremities, stronger on the right side when the dilatation is limited to the ascending portion, and only perceptible under the sternum or ribs when the dilatation is very great. 2. A harsh murmur (rasping sound) synchronous with the pulse, short, commencing and terminating suddenly, perceptible above the two clavicles, and better above the right clavicle, when the dilatation only affects the ascending portion. This murmur extends ordinarily to the back part of the chest. 3. A vibratory tremor, which is best perceived above the clavicles, and in some circumstances below, when the aneurism principally affects the ascending portion. In this last case, the vibratory tremor is always perceptible above the clavicles, especially above the right one.

General Symptoms. — They may be completely wanting. When

they exist they are never very intense unless there be complication of organic disease of the heart.

B. *Sacciform Aneurism.* — 1st. *Sacciform Aneurisms of the thoracic aorta. — Local Symptoms.*—1. Pulsations perceptible above and below the clavicles, but always stronger below. When the aneurism occupies the ascending portion of the aorta, it is under the sternum or to the right of this bone that the impulse is felt. If the tumour have its seat at the commencement or middle of the arch, it is found below the right clavicle and at the summit of the sternum. The aneurisms which affect the first portion of the descending aorta, produce pulsations above and below the left clavicle. As to the aneurisms of the descending portion, they are too deeply placed for the pulsations to be perceived, unless the ribs have been destroyed, and a sensible tumour is obvious externally. 2. In some cases, a true tumour is found, either in the intercostal spaces, or under the sternum, and sometimes even above the clavicles. This tumour is pulsatile, without any change of colour in the skin; but after a certain time the latter acquires a livid tint. 3. Sacciform aneurisms produce, much oftener than aneurisms from dilatation, arching of a portion of the chest. This ordinarily takes place in the superior portion of the right side or towards the summit of the sternum. 4. At the same time, there is dulness on percussion, coinciding with a complete absence of the respiratory murmur in this point. 5. A rough but feebler and less rasping murmur, than in aneurism from dilatation. It is sometimes better heard on the side opposite to that which is the seat of the tumour; and it is often perceptible behind, sometimes better on the left side of the spine than on the right, which indicates that the affection is principally in the descending aorta. 6. A vibratory tremor, best heard above the clavicles, very slightly below, unless there exist an appreciable tumour externally. This tremor, always feebler in sacciform aneurisms than in those from dilatation, often disappears in old and large aneurisms which are obstructed by clots.

Among the *general symptoms*, the most frequent are, a sense of constriction in the trachea, with hissing respiration or hoarseness, and extinction of voice; difficulty of deglutition; pains, varying in their seat, sometimes occupying the vertebral region, and there producing a true terebration, sometimes radiating to the left shoulder, arm, and forearm, of the same side, and accompanied by numbness and partial paralysis in this member; at other times, consisting only of a feeling of weight or fulness about the chest. There are also swelling of the veins of the base of the neck, differences between the two pulses, and sometimes symptoms which belong to organic diseases of the heart.

2d. *Sacciform aneurisms of the abdominal aorta. — Local Symptoms.* — 1. Tumour in the abdominal cavity, presenting pulsations of extreme violence, accompanied by a strong rising movement. By the application of the stethoscope, it is found that the lateral dimensions of the tumour exceed those of the natural aorta, that the

lateral expansion is generally equal, or nearly equal, to the expansion from behind forwards, and that this tumour, somewhat rounded, is fixed in the part which it occupies. 2. Dulness on percussion, provided the tumour be superficial, and has at least the size of an apple. 3. A blowing murmur, short and abrupt, which never presents the roughness of that of the pectoral aorta. This murmur is synchronous with the arterial diastole, and generally has its maximum of intensity on a level with the opening of the sac. It may be wanting, and it suffices, in some cases, as Dr. Corrigan has proposed, to cause the patient to lie in the horizontal posture; his head low and feet elevated to make it reappear.

The *general symptoms* are often of no account. When they do exist, they consist in various disorders of the digestive functions, most frequently in œdema of the extremities.

Diagnosis. — The diagnosis of aneurism of the aorta, and of its several varieties, is always enveloped in great obscurity. When, however, we find appreciable pulsations, dulness on percussion, and a rough, abrupt, and short murmur, there are great probabilities in favour of the existence of this aneurism; and these are transformed into certainty when the tumour is subjected to movements of general expansion.

Aneurisms of the thoracic aorta should be carefully distinguished from *diseases of the heart*, which latter may give rise to analogous symptoms, also from tumours of different kinds which may be the seat of pulsations, and from aneurisms of the brachio-cephalic trunk, and of the carotid and subclavian arteries at their origin. When he heart becomes larger, its impulse increases, and the pulsations which result from it may be mistaken for those of an aneurism, if they occupy the præcordial region, and especially the apex of the heart; whereas in aneurism, the pulsations commonly have their seat beyond the cardiac region, along the course of the aorta, and then we have two centres of movement, the tumour and the heart. In *regurgitation* through the aortic valves, which is often complicated with increased size of the heart, some symptoms are met with that relate to the aneurism, such as a constant, well-marked murmur above the clavicles. But this sound is less harsh; the arterial impulse which accompanies it is bounding; and, besides, the physical signs of this disease are present. *Tumours*, which are the seat of pulsations, when they occupy the thoracic cavity, are not generally accompanied by a murmur nor impulse, nor by tremor above the clavicles; the circulation presents no irregularity, and if any exist, it never bears a relation to the apparent extent of the disease. When these tumours are situated in the cervical region, the diagnosis becomes easier; they rarely give rise to anything more than a feeble whizzing; the pulsations and sounds are entirely limited to the side affected; and it is sufficient to touch the tumour to be assured that it has no lateral expansion. Finally, in displacing it from its position over the subjacent artery, the pulsations and murmurs are found to cease.

It remains for us to speak of the *aneurisms* of the great vessels which arise from the arch of the aorta. But we find in these circumstances, along the course of the respective arteries, pulsations with murmur and tremor, perfectly circumscribed and limited to the affected side. These signs are always superficial and very distinct. In some cases, however, it is very difficult to distinguish an aneurism of the aorta from aneurismal tumours of these arteries; when, for example, the aortic aneurism, instead of being formed in the chest, is placed in the sub-clavicular triangle, becomes developed there, and is not accompanied by any murmur or tremor, as we have lately seen.

The diagnosis of aneurisms of the *abdominal* aorta is perhaps environed by still greater difficulties. It often happens that only a part of the signs which characterise this affection are found. We must then only give a probable diagnosis, and wait and observe.

All tumours of the abdominal cavity, whatever be their nature (cancer of the stomach, hypertrophy of the liver, tumours of the pancreas, mesentery, omentum, of the transverse arch of the colon, or of the diaphragm, accumulation of indurated fecal matters, intestinal concretions, masses of worms), may, when situated over the aorta, transmit the pulsations of this artery and cause a murmur: but these are comparatively feeble compared with those of aneurismal tumours, and they never exhibit any lateral expansion. Finally, their relations with the aorta may be made to cease, and it will be found that the artery has not increased in size. Hypertrophy of the liver is recognised by an increased dulness in the hepatic region. Tumours of the colon, stomach, and epiploon, are superficial and almost always moveable. Those formed by collections of fecal matters disappear by the use of purgatives. *Aneurisms of the cœliac trunk* and its branches, and of the superior mesenteric artery, are next to be considered. They are distinguished from aneurisms of the aorta, by the superficial character of the impulse and murmur which accompany them, by their facility of being displaced, and by our readily ascertaining that the aorta retains its natural size. The antecedent and general symptoms of the disease will be advantageously made use of in diagnosis. For example, if the patient is subject to a cancerous cachexia, there will be much probability of the existence of a tumour of the same nature as in other cases of this diathesis. Sometimes there are pulsations perceived along the course of the aorta (*nervous pulsations*), which may cause doubts in the minds of inexperienced physicians. To avoid all error it will be sufficient to remark, that the pulsations are limited in a transverse direction; that the impulse, instead of being gradual, is sudden; that the murmur when it exists is very short, and is perceived along the whole course of the vessel, instead of being circumscribed to one point; and finally, that the patient manifests all the signs of an anemic condition, and of great nervous irritability.

Progress, duration, terminations, and prognosis. — The progress

of the aneurism is commonly very slow. For a long time, the tumour is concealed, nor does the patient himself perceive it. It is only when it has acquired a certain size, and especially when it has caused cardiac disturbance, that dangerous general symptoms are seen to supervene. As a general proposition, we may say that its progress is subordinate to the resistance of the parts surrounding the aneurism. The duration of this disease is generally long, and depends on its form, the situation of the tumour, and the kind of life of the patient. It is not rare to see patients live for a number of years with an aneurism of the aorta, and especially with aneurism from dilatation. Its duration may, however, be short. Aneurisms, for example, which arise immediately above the left ventricle, deprived as they are of cellular membrane, burst very soon into the pericardium. The aortic aneurisms ordinarily terminate by rupture at the end of a shorter or longer time. In some fortunate and very rare cases, the spontaneous cure of these diseases is observed. The rupture takes place by means of an eschar, or by a rent, according to the nature of the tissue which is perforated. The skin and mucous membranes exhibit an eschar, whilst the serous membranes, on the contrary, rupture by a fissure or rent. Aneurisms of the pectoral aorta may open into the bronchiæ, trachea, or œsophagus, the left cavity of the pleura, the posterior mediastinum, or right ventricle,* and the pulmonary artery.† Those of the abdominal aorta may open into the abdominal viscera, — the intestines, the bladder, &c., into the cavity of the peritoneum, or the vertebral canal, and, according to some, into the sub-peritoneal cellular tissue, &c. A spontaneous cure is rare in aneurisms of the aorta.

Treatment. — To give to the artery its natural elasticity and to reduce it to its ordinary size, to cause the formation of fibrinous concretions in the interior of the sac, when the vessel cannot return on itself, to prevent by all possible means the rupture of the tumour, are the indications which the practitioner should propose to himself in the treatment of aneurism of the aorta. The resources of art are most frequently insufficient to impart to the arteries their lost elas-

* The following are the signs of this rupture : *Local Signs*— 1st, a strong, rough and superficial murmur, joined to a continuous rumble. These sounds are most audible about the edge of the fourth rib near the sternum, and thence along the track of the pulmonary artery up to the space comprised between the second and third ribs; 2d, purring tremor at the same points; 3d, feebleness, and extinction of the second sound near the clavicles. *General Signs* — 1st, a very jerking pulse; 2d, considerable, rapid, and universal dropsy; 3d, a livid, as it were, cyanosed colour of the face. All these symptoms commonly supervening suddenly after violent exertion. (Hope.)

† These are the signs of this last rupture : *Local Signs* — 1st, a very loud, sawing, and superficial murmur, prolonged continuously from the first to the second sound, having its maximum along the course of the pulmonary ; 2d, a purring tremor in this artery and in the second intercostal space; 3d, a feebleness of the second sound at the clavicles. *General Signs* — 1st, the jerking pulse; 2d, rapid and general dropsy ; 3d, a cyanosed colour of the face; 4th, the development of all these accidents after a strong muscular effort.

ticity; and they are especially unsuccessful when rupture of the membranes of the vessel has already taken place. To fulfil the first indication, the causes diminishing the elasticity of the arterial tissue must be removed. If these be entirely local, and result from an organic alteration of the walls, they should be treated by direct antiphlogistics, and especially by powerful revulsives, vesicatories, and deep cauteries applied in the vicinity of the tumour. If, on the contrary, the case depend wholly in the increased impulse of the blood forced out by the left ventricle, all those means must be employed which diminish the energy of the contractions of the heart, and the quantity of liquid which this organ projects at each moment into the circulatory torrent. These are the same means which are had recourse to in order to bring about the formation of clots. They consist of absolute repose of body and mind, bland nourishment, in small quantity, the employment of digitalis, which should be suspended from time to time, and especially recourse to sanguineous depletions. Bloodletting, employed according to the method of Albertini and Valsalva, has had, in the treatment of this disease, a reputation which is not now conceded to it. By means of repeated bleedings and strict diet, Valsalva reduced his patients to such a state of feebleness that they could scarcely lift their arm from the bed. After he had drawn as much blood as he thought proper, he diminished daily the quantity of nourishment and drink so far as only to give his patients but half a pound of pudding in the morning, and one-half less in the evening, and nothing else except a very small quantity of water; he afterwards allowed them drink and nourishment by degrees. This treatment has had its vogue, and numerous failures will not be wondered at, if it be considered that, by thus diminishing the quantity of the blood, this fluid is altered, and is deprived of the power of coagulating; and that, by plunging the patient into a state nearly of anemia, the pulsations of the heart are accelerated and acquire extreme abruptness. Finally, in the case where a concomitant disease of the heart exists, this treatment may cause a prolonged syncope, and death itself. The treatment of Albertini and Valsalva is, therefore, dangerous; it is at the same time so severe that it is difficult to meet with persons who will long submit to it. Bloodletting, to the extent of from 16 to 20 ounces, repeated at more distant intervals, as from fifteen days to a month, and to a still greater extent, and at shorter intervals if the patients are more robust, more plethoric, and repair these losses with more facility, will perfectly fulfil the end in view.

We may propose, as mentioned in another place, the treatment which we have already indicated in hypertrophy, for aneurism of the aorta. It will be more or less required according to the nature of the tumour. It should be very active in mixed or false aneurism (whose seat is most generally in the descending thoracic and the abdominal aorta), because sometimes the cure is obtained by the formation of fibrinous clots: it may be much less rigorous in aneurism from

dilatation, the progress of which is not so rapid. This treatment should be continued for one or two years; and when we are led to suppose that the patient is cured, he should be told to keep in a state of repose for one year at least.

When the aneurism of the aorta projects externally, a new indication is presented: the tumour must be guarded against the impulse of external agents, at the same time that, by means of cold applications an astringents, we should endeavour to procure coagulation in the interior of the sac. When it is necessary to support the tumour, it may be done with a belladonna plaster; finally, when it is enormous, and a rupture is feared, it may be protected by a metallic envelope, or by caoutchouc.

CHAPTER XI.

ANEURISM OF THE PULMONARY ARTERY.

Aneurism of the pulmonary artery is excessively rare, and the only variety which is known is aneurism from dilatation. This dilatation may increase the internal circumference of the artery five inches.

The symptoms which indicate its existence are the following: — *General Symptoms.* — 1st, pulsations with vibratory or purring tremor between the cartilages of the second and third left ribs, decreasing from above downwards, but *not perceptible above the clavicles;* 2d, a slight arching at this point; 3d, a harsh and excessively strong sawing sound, which is heard above the clavicles, and over all the præcordial region, and whose maximum is on the prominence between the second and third ribs.

General Symptoms. — Those of hypertrophy with dilatation of the heart, which generally accompanies this morbid state.

Dilatation of the pulmonary artery can only be confounded with dilatation or with sacciform aneurism of the aorta; but never, in either of these cases, do we find pulsations between the second and third ribs. Even if an aneurismal tumour of the arch of the aorta should have destroyed the cartilages of the second and third left ribs, it would not be possible to make a mistake in diagnosis, because the aneurisms of the aorta are very large, and the murmur which they produce is grave and distant, instead of being acute and superficial; and, besides, the tremor would be perceived above the clavicles, which is never the case in dilatation of the pulmonary artery. Nothing is known respecting the causes, progress, duration, prognosis, and treatment of aneurism from dilatation of the pulmonary artery. It is probable that all we have said respecting the dilatation of the aorta is applicable to that of the pulmonary artery.

CHAPTER XII.

MALFORMATION OF THE HEART.

Malformations of the heart are ordinarily congenital imperfections in the structure of the organ, which consist in a want of development of certain parts, or rather in an anomalous configuration of some of them. It would be difficult for us to include in this classification all the known species of vices of conformation, because two are continued with two, or three with three, so as to render the classification almost impossible. We shall describe in a few words, beginning with the most considerable, the congenital alterations that have been hitherto observed.

1. The heart is simple (like that of fishes), consisting in an auricle and in a ventricle, whence is detached a trunk, which divides into the aorta and pulmonary artery. Children affected with this malformation commonly die in a few days after their birth.

2. The heart is composed of two auricles and of a ventricle (like that of the reptiles). A person has been known to live twenty-two years with this vicious conformation.

3. The foramen of Botallus (foramen ovale) remains. This malformation, which is very common, has been observed in the most advanced age.

4. The foramen of Botallus and ductus arteriosus remain, and are both quite permeable. Sometimes, at the same time, the pulmonary artery is obliterated at its origin. The cavity of the right ventricle has been seen to be almost entirely effaced; and in both these cases a perforation of the septum of the ventricles exists.

5. The septum of the ventricles is open: the aperture is narrow, and although near the origin of the aorta, it does not communicate with this artery. Sometimes, simultaneous with this, the pulmonary artery is contracted, and the foramen of Botallus is open.

6. The septum of the ventricles is entirely wanting, and that of the auricles is incomplete.

7. The right auricle opens into the left ventricle, instead of opening into the right ventricle; the foramen of Botallus remains, and the ventricles communicate with each other by an opening situated immediately below the valves of the aorta.

8. The aorta arises at once from two ventricles; that is, the septum of the latter is wanting at the valves of the aorta; by which a free communication is established between the two ventricles and this vessel. In connection with this vicious conformation, there is most frequently found contraction of the pulmonary artery; sometimes a continuance of the foramen of Botallus, and even an alteration of the pulmonary artery, with permeability of the ductus arteriosus.

9. The aorta arises from the right ventricle, and the pulmonary

artery from the left ventricle, and the foramen of Botallus and even the ductus arteriosus remain.

10. The pulmonary artery arises from both ventricles and the foramen of Botallus remains; the descending aorta is supplied entirely by this artery, whilst the ascending aorta follows its usual course.

11. The arch of the aorta is double.

12. The foramen of Botallus closed in the fœtus.

13. The valves of the heart, and especially the valves of the right side of the heart, present congenital vices of conformation (contraction or regurgitation, &c.).

These diseases are complicated with anomalous communications between the cavities of the heart, contraction of the arterial trunks, and of the corresponding cavities.

Of all the vices of conformation, of all the causes which give rise to a communication between the two sides of the heart, the most common is a persistent opening of the foramen ovale, depending either on the valves which close this opening not having contracted adhesions (which is most generally the case), or on their non-development. In this last case the opening may be dilated, and be even an inch wide. This malformation is congenital; but the appearance of all those accidents which indicate a communication between the right and left cavities, supervening immediately after a fall or violent exertion, induces us to believe that there might be, after birth, rupture of the membrane which covers the opening, or separation of the folds that close it.

The mixture of the arterial and venous blood is almost an inevitable consequence of a communication existing between the two cavities of the heart, whatever be the cause. There may be, however, exceptions; as, for example, when the two folds of the valve have not contracted adhesion, their approximation by the pressure which is made against them on both sides is sufficient to prevent the mixture of arterial and venous blood. We can understand, also, although the case is purely hypothetical, that where the opening is large, the blood may make on each side of this opening an equally opposing pressure.

The communication of the right and left cavities of the heart has for its most frequent result *hypertrophy with dilatation* of the right cavities. Can it be true, as MM. Bertin and Bouillaud think, that hypertrophy results from the introduction into the right cavities of a certain quantity of arterial blood, which is endowed with more irritating and nutritive qualities than the venous blood? We do not deny the influence of this cause; but if it be considered that the malformations of this nature, in which hypertrophy of the right cavities is at its maximum, are those in which contraction of the pulmonary artery coincides with the continuance of the foramen ovale, we arrive at the conclusion that the penetration of arterial blood is only one of the least of the causes of the hypertrophic development of these cavities, and that the most powerful

cause is found in the contraction of the orifice which almost always accompanies the anomalous communications between the cardiac cavities. Will it be objected, that in the case of contraction of this orifice, the anomalous opening is abundantly sufficient to allow the flow of blood? We reply, that it cannot be doubted that the ventricle empties itself with more difficulty through an artificial opening, than through a natural one. The cause of the hypertrophy of the cavities, in the case of communication between the cavities of the heart, is indirectly owing to the contraction of the pulmonary artery. As to the dilatation, it appears to us to be owing to the excessive distension which the right cavities experience, in consequence of the obstacle to the circulation of the blood.

Symptoms. — A. *General symptoms of the communications of the right and left cavities of the heart.* — Blue or violet colour of the skin (*cyanosis**) more marked than in any other disease of the heart; and extending often over the whole body, but more particularly evident at the openings of the mucous membranes, the extremities of the hands, and feet, the pulp of the fingers and of the toes which are swollen, hot, and sodden; considerable depression of temperature; frequent syncopes; sometimes convulsions and great dyspnœa, are the symptoms described by authors. All these signs are certainly observed when the arterial and venous blood is mixed in large proportions, and when the obstructions to the circulation are extreme; but when this is not the case, the general signs are no more than those belonging to simple disease of the valves. It becomes necessary, therefore, to have recourse to the physical signs of the disease, and to the antecedent symptoms, in order to form a diagnosis.

B. *Local Symptoms.* — Augmentation of dulness on *percussion* indicates hypertrophy or dilatation of the ventricles: increase of the *impulse* below the sternum indicates hypertrophy of the right ventricle. *Auscultation* shows an excessively *strong* murmur, which is *superficial* and *near*, and whose maximum is found immediately above the semilunar valves, or on a level with the inferior border of the third rib. If this murmur depends on a contraction of the orifice

* Is cyanosis the result of the admixture of red and black blood, as Corvisart and Gintrac have asserted? Or does it not rather depend on the obstacle which the diseases of the orifices, and of the muscular substance of the right side of the heart opposes to the venous circulation, as Morgagni, M. Louis, Hope, &c., thought? This question is the more difficult to decide, since there is rarely any communication between the right and left cavities of the heart without diseases of the orifices or of the muscular substance. But, as only a small number of observations exists in reference to communications without any well marked alteration of the circulatory apparatus, in which cyanosis did not exist, or was slightly marked, we think that this particular colour of the skin is due more to an obstacle to the passage of the blood than to the mixture of the two varietes of blood. We are not the less disposed, however, to believe that this admixture has some agency in the development of the phenomenon, because in diseases of the valves of the right side of the heart, however much advanced they may be, there is never found so violet a colour of the skin, except when there exists an anomalous communication between the two sides of the heart.

of the pulmonary artery, it extends along the course of this vessel, as far as on a level with the second intercostal space, accompanied by a tremor. When it results from a communication between the right and left ventricle, it is better heard along the course of the aorta than along that of the pulmonary artery; and when it is produced by this double lesion (the contraction of the pulmonary orifice and communication between the ventricles), it extends along the course both of the aorta and pulmonary artery.

Diagnosis. — When the preceding signs occur in conjunction with cyanosis, there can be little doubt respecting the existence of a communication between the right and left cavities of the heart. The presence of hypertrophy of the left ventricle adds still more to its certainty. When, on the contrary, no cyanosis is found, recourse must be had to the antecedent symptoms. If, from his earliest infancy, the patient has presented the symptoms of an organic disease of the heart, especially if he has never had endocarditis, to which alteration of the valves could be referred, it is very probable that there is malformation of the heart, consisting in a communication between its right and left cavities. Care must be taken not to confound the murmur which is heard under these circumstances with that accompanied by tremor, which is heard in the case of dilatation of the pulmonary artery. This last murmur will be distinguished by having its maximum in the second intercostal space, instead of having it on a line with the valves.

THIRD CLASS.

NERVOUS DISEASES OF THE HEART.

Among the diseases which are incident to the dynamic functions of the heart, we have only to study *palpitations* and *syncope*. The *spasm* of the heart which Laennec has described, is generally regarded as an imaginary disease.

CHAPTER I.

PALPITATIONS.

Palpitations consist in an increase of the force and frequency of the pulsations of the heart, so as to render them perceptible, and even disagreeable to the patient. They vary in force, from a degree

in which they are hardly sensible to one of great violence. The patients often hear the pulsations, especially when they lie on the left side; even the two sounds of the heart may be distinguished in this posture. During the palpitations, the sounds of the heart ordinarily increase in intensity; and, in some circumstances, a slight bellows sound is produced, which ceases and disappears when the circulation has regained its natural quietness. Palpitations are also accompanied by a slight peripheric friction sound, which is dry and superficial, and due to the rapidity with which the two folds of the pericardium glide on each other: it disappears with the cause which originated it. At the same time, the arterial diastoles are short, and quick, and the veins of the lateral parts of the neck are swelled and distended. Palpitations, when prolonged, always cause a sensation of uneasiness and anxiety in the præcordial region, and of fatigue in the whole extent of the chest; also, quickness of breathing, sometimes even extending to syncope.

Palpitations form two great classes; viz., those which are caused by a material alteration of the heart (*palpitations from organic cause*), and those which arise from a nervous cause (*palpitations from inorganic cause*), and are not referrible to any material lesion of this organ. *Palpitations from organic cause* are distinguished from the others by their permanence and tenacity, by the great irregularity which they cause in the pulsations of the heart, and in the sounds, which are considerably feebler than common, and finally, by the existence of the signs peculiar to the affection of the heart which has produced them.

Nervous palpitations, of which only we intend to speak here, constitute a very troublesome disease. They present many varieties which it is important to distinguish, because their treatment is different, and sometimes even opposite. Everything else being equal, they are more violent in proportion as the patients have a more nervous constitution or a more irritable temper.

In the *first variety* we shall place palpitations induced by strong mental emotions, by hysteria hypochondriasis, nostalgia, excessive labour, particularly in the night, venereal excess, dyspnœa, &c. These are nervous *palpitations, properly so called.*

In the *second variety* we place palpitations produced by the abuse of spirituous drinks, and by exciting regimen, and especially by plethora (*plethoric palpitations*).

The *third variety* will consist of palpitations which appear in individuals who are anemic and chlorotic, and which are owing to an impoverishment of the blood (*anemic palpitations*). These last palpitations are very often confounded with palpitations from organic causes, although it is easy to distinguish them by a close examination; for all the characters of anemia and chlorosis are present. We assure ourselves that the heart has not increased in size, that its orifices are in their natural state, and that the valves are completely closed. In speaking of the intra-cardiac murmurs, we have already given the means of distinguishing murmurs by organic

causes from the murmurs by inorganic causes. We shall not repeat them here. The case most difficult to be recognised would be that where palpitations from organic and inorganic cause existed at the same time.

Progress, duration, termination. — The progress of palpitations is always irregular: they ordinarily return in paroxysms at more or less distant intervals. As to their duration, they vary according to a number of circumstances, and especially according to the cause which has produced them. It is difficult to fix a termination to so transient an affection. It may, however, be said, that when they are prolonged, or are repeated for a long time, they finish by causing hypertrophy or dilatation of the heart.

Treatment. — The treatment of palpitations is entirely subordinate to the causes which have produced them; and to the ascertaining of these must the efforts of the physician be directed. Palpitations from moral causes can only be managed by moral treatment. This last is not so successful against palpitations produced by hysteria or nostalgia. As to those which are produced by dyspepsia, the means which are commonly directed against this affection of the digestive canal should be employed. Plethoric palpitations require a more active treatment. Sanguineous depletions, moderate regimen, exercise, are the means which succeed the best. In anemic palpitations, tonics should be had recourse to, as well as the chalybeate preparations, cold baths, nourishing food, moderate exercise. The effects of chalybeates may be considered as the test of the character of these palpitations. If they do not succeed, an organic affection is to be feared. The preparations and mode of administration must be varied. Among those of iron we place, in the first rank, the subcarbonate and lactate of iron.

CHAPTER II.

SYNCOPE.

Faintness, lypothymia, syncope, are the three stages of a similar affection of the heart, which consists in a feebleness of the innervation of this organ that may even extend to a complete suspension of its contractile power.

A variety of causes may occasion syncope; viz., moral emotions, a feeling of disgust, physical pains, certain colours, shocks of the nervous system, sudden abstraction of a large quantity of blood; passage from the horizontal to the vertical position when the patients are anemic; great obstacles to the circulation, which act by causing congestion in the heart; the action of certain poisons, as hydrocyanic acid, of certain miasmata, and, finally, all the causes, which

directly or indirectly suspend, for a few moments, the irritability of the heart.

There is no need of enumerating the well known phenomena of syncope: they are those of sudden death, with this difference, that in the majority of cases the individual may be recalled to life. The ordinary duration of syncope is from a few seconds to a few minutes. It may, however, last for a whole hour. The circulation is seldom entirely arrested: the pulsations of the heart are not entirely suspended; they are only very feeble, but the second sound is always perceptible. Whilst the sounds of the heart are audible, there is hope of restoring the patient to life.

Syncope, which, when it is nervous, does not present any danger, becomes alarming when it coincides with an organic disease of the heart, or with anemia, because sudden death may be its consequence.

Syncope, when purely nervous, requires no other treatment than the horizontal posture, fresh air, the quick dash or sprinkling of cold water, and stimulation of the pituitary membrane with Cologne water or ammonia. That which is symptomatic of another disease, as that of the heart, for example, requires, independently of the preceding measures, the employment of the treatment appropriate to it. Finally, anemic individuals should be constantly kept in the horizontal posture, until the tendency to syncope has disappeared.

APPENDIX TO DISEASES OF THE HEART.

CHAPTER I.

HYDRO-PERICARDIUM, HEMO-PERICARDIUM, AND PNEUMO-PERICARDIUM.

Every effusion of abundant serosity originally developed in the pericardium constitutes *hydro-pericardium.* These effusions frequently appear as a consequence of general dropsy, or of an acute or chronic inflammation; but they are rare, as an idiopathic disease. They may be the result of an exhalation which takes place in the last period of life. In general dropsy, the pericardium always contains, in proportion, less liquid than the other serous cavities. It is rare to find more than sixteen ounces, although Corvisart is said to have found eight pints. The liquid which is therein enclosed

is ordinarily yellowish and transparent; it is only troubled and flocculent when there is pericarditis.

The *general symptoms* which authors assign to pericardic dropsy are very obscure; viz., weight in the region of the heart, a floating sensation of the organ which patients experience, undulations of the liquid heard and even seen between the intercostal spaces, irregularity of the heart's pulsations, frequency and intermission of the pulse, orthopnœa, palpitations, and syncopes. All these signs have nothing very characteristic of the disease.

The *local symptoms* allow us to recognise with facility these effusions when there is from ten to fourteen ounces of liquid in the cavity. The dulness is increased, and ascends under the sternum as far as the second rib: there is found in the præcordial region a notable *arch;* the *impulse* is feeble, undulatory, *variable in its seat;* the *sounds*, especially the first, are distant. Hydro-pericardium, which is the consequence of a general dropsy, requires the same treatment as dropsy; that following inflammation of the pericardium should be treated in the same way as pericarditis. It is in the treatment of pericardic dropsy which is thought to be idiopathic, that the operation, which consists in puncturing the pericardium, has been proposed. To be consistent with themselves, they who proposed this operation should use irritating injections into the pericardium, in order to cause an intimate adhesion of the walls. These injections have also been proposed; but up to the present time, the whole is merely a project, and a prudent practitioner would abstain from the performance of operations so dangerous and so unreasonable.

The effusion of blood into the pericardium, or hemo-pericardium, has various causes; it may be the result of the rupture of one of the vessels of the heart itself, or of the great vessels which arise from it. The blood effused in larger or smaller quantity, is commonly in a great degree coagulated. The physical signs of this effusion are the same as those of serous effusion.

Finally, a certain quantity of gas may be found in the pericardium (pneumo-pericardium). Little is yet known respecting the quantity, quality, or chemical nature of the gases which are met with in the pericardium. Most frequently it exists in conjunction with an effusion of liquid. The cardiac region is then resonant on percussion, and a sound of fluctuation, caused by the pulsations of the heart, is heard. M. Bricheteau has noticed the existence of a sound which he has compared to that made by the wheel of a mill.

When an incision is made into the pericardium, the gases escape with a hissing noise. Laennec thought, but without any foundation, that the propagation of the sounds of the heart to a certain distance along the walls of the chest was owing to the instantaneous development of gas. It is most often reabsorbed, and its presence in the pericardium does not give rise to any grave accident.

CHAPTER II.

POLYPI OR SANGUINEOUS CONCRETIONS IN THE HEART.

Under the head of polypi, we comprehend principally the sanguineous clots which are formed in the heart during life. That the blood may coagulate in this way is an incontestable fact of familiar observation, since it is thus found in the arteries and veins during life, where it becomes organized, contracts adhesions with the walls, and finishes by obliterating the caliber of the vessels.

It is in the right cavities, in the auricles especially, that polypous concretions are most often observed ; doubtless, because the blood stagnates more easily in these cavities, and accumulates in larger quantities there, during the last moments of life. Perhaps, also, as M. Bouillaud thinks, this disposition should be attributed to the frequency of venous inflammations, which extend sometimes even into the right cavities, and to a more marked tendency in the venous blood than in the arterial to coagulation.

Anatomical Characters. — 1. *Inorganized polypi.* The polypi which form after death, or in the last moments of life, are composed of black blood about half coagulated, covered to a greater or less extent by a layer of yellowish fibrin, of variable thickness. Sometimes they consist in a yellowish or whitish shining mass, transparent as jelly. These concretions do not adhere to the walls of the heart, nor present any trace of organization, and they are principally met with in the right cavities.

2. *Imperfectly organized polypi.* — Polypi which have begun to be organized before death, are more firm, more opaque, less loaded with serosity, and of a more distinctly fibrous texture, than the preceding. They are often arranged in concentric layers; are whitish or yellowish, exhibiting in some points a tint which is the colour of flesh or slightly violet, and adhere strongly to the walls of the heart. This adhesion often takes place through the medium of a filamentous tissue, whose rupture leaves rugosities on the internal membrane of the heart, and on the surface of the polypus : their surface is often studded with bloody spots, of more or less depth. Some polypi contain, in their interior, pus of a healthy nature, or pus which is concrete and sanguineous, causing them to resemble the *globular vegetations* which we have described in the chapter on *Diseases of the Valves.*

3. *Polypi completely organized.*—These are polypi of old formation, and sometimes many months before death. They are entirely opaque, and resemble perfectly the oldest clots enclosed in aneurisms. Their adhesion to the walls of the heart is extremely solid ; they are met with as often in the left as in the right cavities.

Causes. — The development of the polypous concretions of the

heart may take place under the influence of *mechanical causes*, or of *vital causes*.

1st. *Mechanical causes.* — Everything which creates a mechanical obstacle to the course of the blood, and causes its stagnation (diseases of the valves, dilatation, softening of the heart, cachectic diseases, approaching death, &c.), may produce sanguineous concretions of the heart, and give rise to the exudation of plastic lymph and the formation of adhesions, but only when the life of the individual is prolonged for a certain length of time.

2d. *Vital causes.* — Inflammation of the internal membrane of the heart, introduction of deleterious substances into the circulation, the particular tendency which the blood has to coagulate under the influence of a phlegmasia already existing, are the principal vital causes of the polypous concretions of the heart.

Symptoms. — A. *General Symptoms.* — Sudden and great increase of dyspnœa; small, feeble, irregular, unequal, and intermittent pulse; intolerable feeling of anguish and suffocation; sleeplessness; continual agitation; coldness of the extremities; livid colour of the face; nausea and continued vomitings, and in some cases stupor and feeble convulsive movements.

B. *Local Symptoms.* — The pulsations of the heart become extremely confused and irregular; the dulness on percussion increases when the polypous concretion is so excessive as to distend the cavities of the heart. Auscultation renders evident a notable diminution in the resonance of the sounds of the heart, which become dull and stifled. Under some circumstances, where a polypous concretion is caught by a valve, or hangs freely in an orifice, as in a case which we have seen, a soft murmur is heard, whose maximum varies according to the seat of the polypous concretions and the affected orifice. This murmur can only be considered as a sign of the existence of a polypus, when we can ascertain: 1st, that there did not exist any anteriorly; 2d, that it does not depend on an inflammatory affection of the valves of the heart.

Diagnosis. — When an extreme irregularity of the pulsations of the heart and arteries is observed to supervene, in conjunction with all the general and local symptoms which have just been noticed, there can be little doubt respecting the presence of a polypous concretion in the heart. The difficult cases are those in which there existed already a disease of the heart, with irregularity in the beats of this organ. Even, in these last cases, if the irregularity be suddenly aggravated, if the beats become abnormal, more confused, more obscure, if all the preceding general symptoms are found, it may be almost always affirmed that a polypus of the heart exists. The polypi which are formed a long time before death are very far from being so easily recognised, especially those which are so limited as to give rise to the symptoms of a disease of the valves. We confess that we have no other means of resolving this difficulty than by reference to those drawn from the progress of the disease and from the mode of connection of the symptoms.

Progress, duration, terminations, and prognosis. — Sanguineous concretions once organized generally continue till death, and do not diminish in size. Can recent and small concretions disappear by absorption, as M. Bouillaud thinks? This is probable, but is not proved. Polypi of the heart commonly terminate by death in a very short time. Those which occupy the auricles, and especially those which acquire a great size, cause death the most promptly. It thence follows that the prognosis of polypous concretions of the heart is always very unfavourable.

Treatment. — There is nothing more unsettled than the treatment of polypi of the heart. M. Bouillaud proposes bloodletting to be repeated with more or less frequency, in order to modify the tendency which the blood has to coagulate. Hope rejects this practice as dangerous and inefficacious. Finally, other authors, guided solely by theoretical views, propose the use of diluent drinks, given in great abundance, and the salts of potassa, solely with the view of dissolving these concretions. This diversity of opinion only proves that no efficacious treatment against this disease is yet known.

THE END.